Cutaneous Immunology & Allergy

- Immunology of skin
- Eczema (Dermatitis)
- Urticaria & Angioedema
- Drug Eruption
- photosensitivity

References.
- Sarrub
- Darouti
- Andrews
- Balognia
- E. medicine
- Derm net

تراجع مع Rook.

Eczema (Dermatitis)

Introduction:

*Def.
*Incidence
*Pathogenesis
*C/P
*Complications
*Treatment

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* definition:-

. Eczema: Come from the Greek word "Ekzein" (فورانه)
ω means "To Boil" or "To effervesce" (يفور)

. Dermatitis: literally (حرفيا) = inflamm. of the skin (كلمة عامة)
So it's broad (non) specific Term (all Ecz. are dermatitis but not all dermatitis are Ecz.)
But (مش العكس)

Both words used by dermatologist to describe group of inflammatory skin disorders caused by variety of Ext. & internal stimuli That's characterized:

- Clinically by (3) phases (see C/P)
- Histopathologically by (2)
  1. Spongiosis
  2. Exocytosis.

NB

. Eczema: تستخدم اكثر في اوربا
. Dermatitis: " اكثر في أمريكا

Acne Rocasea > [illegible] perioral dermatitis

ما ينفعش
أقول عليها
Eczema.

- Pathology of ECZ. : (باثولوجي)

Perioral eczema = CD
citrus food / Tongue induced.

• Acute ECZ.

- Early (Dermal): - Dermal Edema
  - Perivascular Lympho-histiocytic infilt.
- then (Epid): ⊙ Spongiosis
  - ⊙ Exocytosis
  - ⊙ Blister format^n (Vesicle)

• Subacute ECZ.

- Spongiosis ↓↓
- ⊙ Parakeratosis
- ⊙ Acanthosis
- Crustat^n (Coagulated plasma + Pyknotic Nuclei).

• Chr. ECZ. ← Acanthosis, ParaK., Dermal fibrosis.

- Epidermal → Acanthosis & Parakeratosis.
- Dermis: Dermal fibrosis (detected clinically as Lichenificat^n)
  - ↓ psychic ptn يحصل على طول بدون المراحل الأولى (1ry lichenific...)

NB Exocytosis is (lymphocytes infilt.) + (spongiosis) if there is :-

- ↑↑ Marked spongiosis + Marked Lymph. infilt. → Dermatitis [ECZema]
- ↓ Scanty spongiosis + ↑ Marked infilt. → (MF)

C/P of ECZ. (General Features)

لازم
- Itching (Cardinal)
- Ill defined (DD from papulosq. Erupt^n)
- has ③ phases.

• Acute

- Erythema
- ✓ papulovesiculat^n
- blister & ± bullae
- ✓ Weeping = يبكي
- ✓ oozing

• Subacute

- Erythema
- Papules
- Scaling & Crustat^n (∞)
- (↓) Vesiculat^n & oozing). بيأ يخف

• Chr. (ē ch itching)

↓ بتحرك / مزمنة

Lichenificat^n. Δ
- thickening
- Hyperpigm.
- ↑↑ skin Markings → (Exaggerated)

Treatment of Eczema

Acute Weeping ECZ.

1. drying antiseptic lotions

٣ قطعات ← ٣ مرات

- 10 mins Cool Soaks of
  - → 0.65% Alum. Acetate
  - → saline
  - → Tap Water
  - K-premengnate

then

2. Smear of Cs Cream or lot^

0.65 Al Acetate = purrasz sol

مثال

3. non adhesive dressings

non occlusive

→ Staph موجود عليه

Subacute Eczema

1. Cs lot^s or Creams
2. فيه بالك وجود ← أمس
   yellow crust = Inf → Fucicort

NB • staph Colonize:-
(i) all Weeping ECZ.
(ii) Most of dry ones.

→ to differentiate bet. Clonizat^ & overt Inf:-
(1) Heavy Crustat^
(2) large No. Isolat^
(3) CRP

• staph. Inf. or clonizat^ may aggrevate ECZ. & delays Healing

← See Eczema = Think staph

لازم افكر ĉ staph

Chr. lichenified ECZ

رطب

مرهم

Cs Ointments

also
- Calcineurin inhibitors (CI)
- Icthammol
- Zinc

ولازم تختار Cs قوة على حسب: - age - site - amount

السن . مكان . الكمية

50

نصائح

1. don't use potent [perderm, micort] Cs > Hydrocortisone 1% in (3F)
   - Face
   - Flexures
   - Infants

2. don't use:
   - hydrocort > 200 gm/W of mild potent
   - pento vet > 50 gm/W of mod. potent
   - dermo vet > 30 gm/W of superpotent

أخففه بغاز لبيد عام أزود الكمية

أعطيه 2-3 W then every 4 weekend

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5 Should topical steroids be used on lesions that may be infected or colonized with bacteria?
Absolutely. Lesions get infected because the skin defense barrier is broken. The topical steroids allow it to heal, and thus the bacteria have greater difficulty infecting the skin. Such bacteria have receptors for fibrin or fibronectin that can be exposed in dermatitic, but not normal, skin.

- Acute Weeping ECZ. → drying Antiseptic + Cs lot^ chlot
- Subacute ECZ. → Cs كريم
- Chr. lichenified ECZ → Cs مرهم
  - ↑ potency
  - ↑ moisturizing

Classification of eczema.

According to the causative factor whether external or internal;eczemal can be classified into :

Exogenous Eczemas	Endogenous Eczemas
①-Contact dermatitis (CD): • Allergic CD • Irritant CD • Photoallergic CD ②-Infective dermatitis ③-Dermatophytid = (Id) Reaction. ④-Eczematous PML ⑤-Post-traumatic eczema	✓-Atopic dermatitis -Seborrhoeic dermatitis -Stasis dermatitis -Frictional lichenoid dermatitis -Xerotic eczema -Discoid eczema -Pityriasis alba -Juvenile plantar dermatosis -Eczematous drug eruptions -Metabolic eczema or eczema associated with systemic disease →Hand eczema -Exudative discoid and lichenoid dermatitis -Chronic superficial scaly dermatitis

= genetic

Sun exposure

NB: 1- This classification isnot absolute because of interaction between the 2 factors.

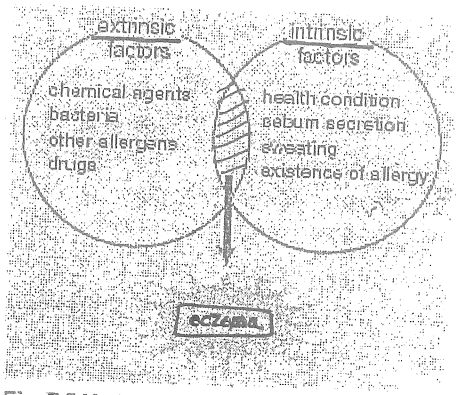


Fig. 7.5 Various factors causing eczema. Extrinsic and intrinsic factors interact, resulting in eczema formation.

Allergic CD (ACD)

Dermatitis Venenata

- def
- Incid
- Pathophys.
- Invs

- **def** delayed Hypersensitivity reaction (Type IV Allergy) resulting from cut. Contact ē specific Allergens to ẅ the patient develop specific Allergy. *
- **Incid** (20%) of CD cases (ICD = (80%), ACD (20%))
- **Pathophysiology**: Type IV Delayed Hypersensitivity react^n ẅ has 2 phases:- (ببساطة)

(1) التعرّف \ (1) Induction (sensitizat^n) phase : (10-14 ds) — 1st exposure (2 wks)

(2) الإثارة (2) Elicitation (challenge) phase : (1-3 ds) — Recurrent exposure (2ds)

[1] chemicals that Can produce ACD:-

- ≃ 3000 in No
- of small sized molecules <500 daltons.

[2] Pathogenesis: LMW Ag hapten <500 daltons → When Contacts the skin form → hapten-Carrier protein Complex → LCs display it as complex with (HLA DR) on their surfaces → presented to CD4 → (ببساطة) Interact ē TCRs CD3 Complex of CD4 →

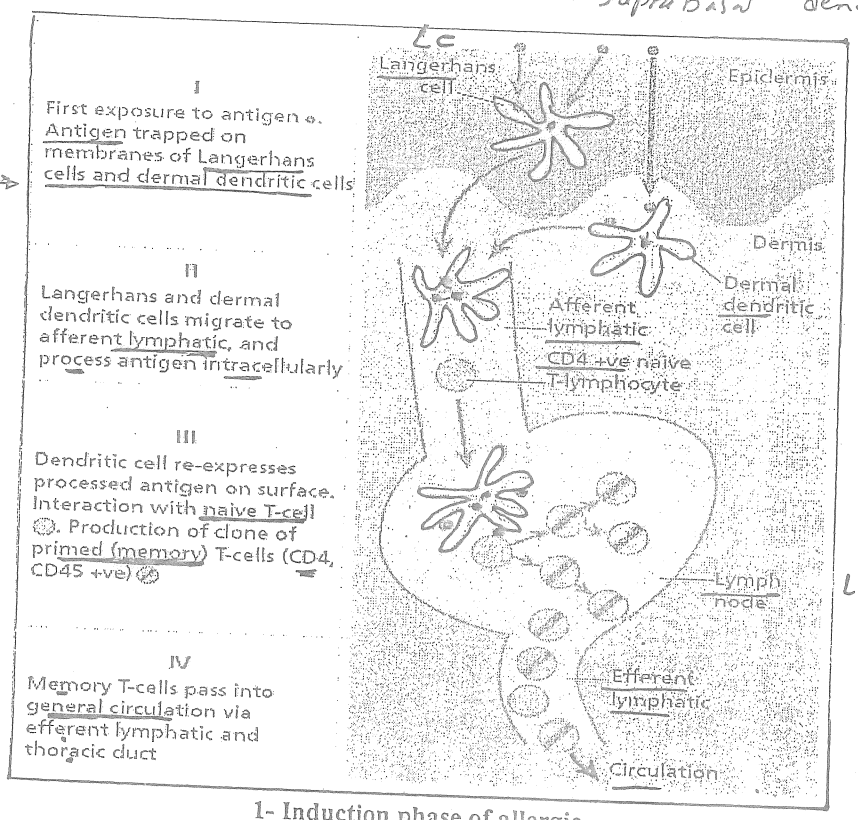
[3] ACD ch^By:

(مميزات)

(a) Previous Contact is Needed to induce Allergy.
(b) (Specific) to (one) chemicals & close relatives
(c) After Allergy has been Established [all] areas of skin will react to the Allergen
(D) Sensitizat^n persist "indefinitely" (طول العمر)
(E) Desensitizat^n ↓- seldom occurs. (نادراً)

Suprabasal dendritic cells

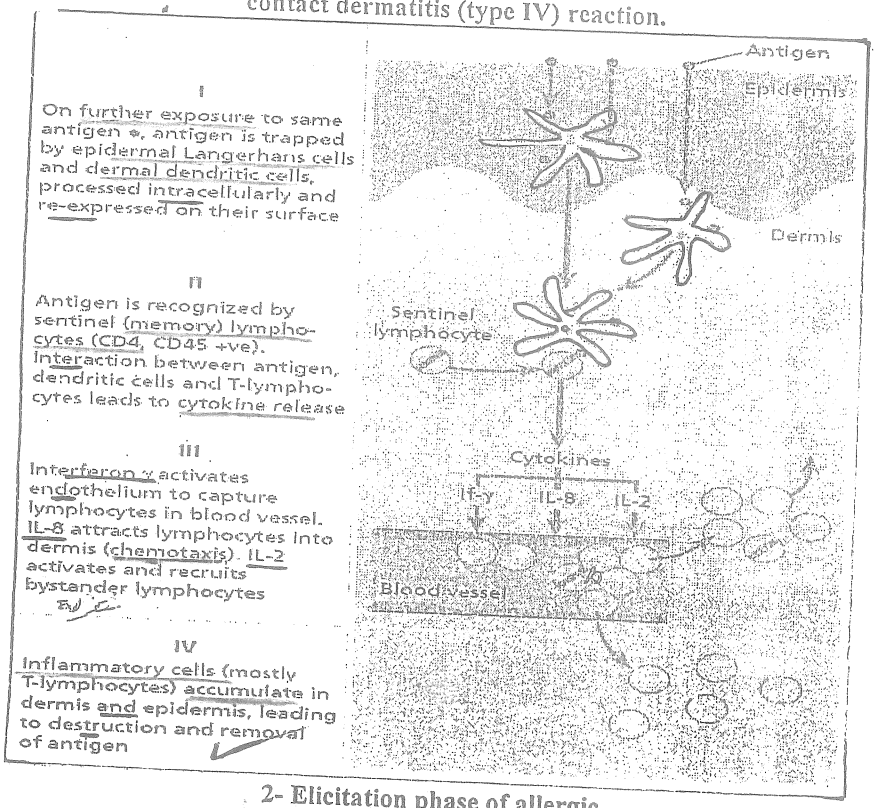
Ag → LCs antigen presenting cells → afferent lymphatic → L.N → efferent → circulation



L.N

1- Induction phase of allergic contact dermatitis (type IV) reaction.

Naive → memory → sensitized → lymphocyte → ACD



2- Elicitation phase of allergic contact dermatitis (type IV) reaction.

تفاعل

IL8 → Chemotaxis
IFN-γ → ++ Endoth. → Capture (TC) in BVs.
IL2 → ++ recruit Bystander L

NB → IL8 → ACD عيزا وليس ICD

ACD ← dermis ال Bl.Vs من lymphocyte خروج

Clinical picture of ACD :-

(A) Clinical presentat^ → onset (البداية), site (مكان), pattern (الشكل) (Q)

(B) Causative Allergens

(C) Regional ACD (Q)

[A]. Clinical presentat^

(1) Classical (Common = Eczematous) pattern

- [1-] Acute weeping Ecz.
- [2-] Subacute Ecz.
- [3-] Chr. lichenified Ecz.

(2) other less common = Non Eczematous ACD (Q) سؤال المقصورة مش شكل ecz

- Urticaria / Purpura
- EM like / L-p like
- Pigmented / depigmented
- Papular / Vesicular / vasculitis
- baboon Synd : deep red- violet Eruption on buttocks, genitalia, inner thighs & ± axillae → SDRIFE: Symmetrical Drug related Intertrigenous & flexural Exanthema. Drugs: Ampicilin, Nickel, Mercury الاسم
- onycholysis
- 2ry disseminat^

Site

مكان → usually [localized] to site of Allergen Contact but [Generalizat^] may occur تنتشر (Morbilliform or Exfoliative or 2ry disseminated ACD) ← Rash Id Reaction

لو من معصم الساعة بياخد نفس الشكل → The initial site of dermatitis often provides the best clue regarding the potential cause of ACD. (See regional ACD)

onset

يوميه عكس اسبوع → Usually 1-3 days from Contact ē specific

Allergen in patient who have previously sensitized by the same Allergen (= Elicitation phase).

باستثناء

Neomycin — سكر كيتاج لفترة — Eg: (Poor Cut Abs)

Cement

NB: Induction phase may be caused by a Topical agent while Elicitation phase caused by the same Agent but when given systemically → ACD w ±:

① localized to site of previous contact or

② 2ry disseminated. Generalized

* This condition called ← **Systemic ACD** → eg Cinnamon oil & other medications.

B. Causative Allergens

→ About (25) chemicals responsible for (50%) of ACD cases → see the table

(≥ 3000 Ag)

→ Commonest of them are: (10)

- Also: Toxicodendrons (Poison Ivy, oak, sumac) ... تاني

is a classic Example of ACD in North America w is ch by (linear streaks) of ACD that develop when plant parts have been in direct Contact ē skin. (Usually: fingers, Wrist forearm, genitalia)

- Nickel.
- Poison IVY
- Fragrance Mix

(أدوية)

- Neomycin ✓
- Bacitracin ✓
- Topical Cs ✓
- Thimerosal

(معادن)

- Gold
- chromate
- Cobalt

- C: Balsam of pera
- Paraphenylenediamine
- C: formaldehyde
- Rubber

Top 10 Allergens Identified by NACDG

(1) Nickel	(6) Gold
(2) Neomycin	(7) Quaterinum 15
(3) Balsam of peru	(8) Foramldehyde
(4) Fragrance mix	(9) Bacitracin
(5) Thimerosal	(10) Cobalt

Note
metals (1,6,10)
Preservatives (5,7,8)
Topical Antibiotics (2,9)
Fragrances (3,4)

+ حاجات
Poison Ivy
Chromate
Rubber
Cs

Nickel الأشهر الجد المعلب المحفوظات Commonest in female	*It and Poison Ivy are the commonest allergens at all(شفوي) ↘ linear stricks امريكا → مع البنات ممكن يبقي موجود في: (١) الاكل: الاسباجيتي المعلب-الفول المعلب- والخضروات والفواكه المعلبة. بيبسي بيبرا - البصل - شاي - الكوكا - المحار - البقول - السبانخ - الشيكولاتة (٢) المعادن: زي: *الحلق(لوكان فالصو اودهب بس الكلبيس نيكل). earings *العقد(الرقبة) neck lace *زرار الجينز(jeans stud). *خرامة الاذن *العملات المعدنية(حساسية علي الجلد اللي تحت الجيب) *ساعة اليد(معصمها او الاطار) hand watch Commonest sites of nickel ACD *Ear lobes.....from earrings or piercing by nickel material. *Neck.....neklaces *Wrist.....watch strap *Lower abdomen.....jeans button or stud *Hand.....pompholyx لاحظ: عشان تمنع حساسية فتحة الاذن مكان الحلق: ١-استعمال خرامة اذن من الاستانليس وليس من النيكل. ٢-ممنوع استعمال الحلقان الفالصو ٣-لازم كلبيس الحلق الدهب يكون خالي من النيكل(دهب او قصدير). ملحوظة: الاكلات التي لايوجد بها نيكل: السمك-اللبن-البيض-اللحمة-البط-القهوة - الخضروات والفواكه الطازجة مفيش شئ
Neomycin + Cs Bacitracin	- Topical antibiotic used in skin, eye, ear and piles preparations, alone or with bacitracin ,Cs, or antifungal eg. (Kenacomb cream.) - Crossreaction with bacitracin, gentamycin, streptomycin may occur.
Balsam of Peru التوابل. المستحضرات	Naturally occurring fragrance used in: Some scented cosmetics, spices and suppositories. نشوفها في المرضي اللي عندهم حساسية من الطماطم-الموالح-القرفةcinnamon-القرنفل-الثوم-الخمرة والسجائر.
Fragrance mix. روائح عطر-شذا عبير	-Most common cause of allergy to cosmetics -Uses: * To give pleasant odor (مكسبات رائحة) ← * To mask unpleasant odor: عشان كده بتبقي موجودة في المستحضرات المكتوب عليها (unscented). -So use fragrance free preparations

	NB: allergy to cosmetics may be due to: fragrance mix, preservatives (Q.15, dyes and lanoline).
Thimerosal ثيمروزال مادة حافظة لمحلول العدسات التطعيمات	Preservative material used in: 1. Vaccines 2. Contact lens solutions 3. Ophthalmic and ID testing solutions 4. Antiseptics and cosmetics (لو واحد بياخد مستحضر للعين او الاذن وحصلتله حساسية منه؟ايه ممكن يكون السبب (2))
Gold "اكثر لو تم استعماله" "في الاسنان"	*Inert material used in jewelry,dentistery,electronics *Rarely considered as a cause of Jewellery dermatitis but this common if the patient is wearing dental gold * presentation:- hand ,facial and eyelid dermatitis -Oral lichenoid eruption
Formaldehyde (colorless gas)	Colorless gas used in: Shampoo, cosmetics, newsprint. deodorant, smoke, car exhaust and clothing (يخليها تنشف بسرعة ويمنع تكسيرها wash and wear, antiwrinkles)
Quatrineum 15 شامبوهات - صابون - الرطبات - مضادات الحيوية والفطريات	Preservative with antibacterial and antifungal properties. - used in shampooes,moisturizers, soaps and cosmetics (commonest preservative)
Cobalt - الجوهرات - الفخار - الطوب - صبغات الشعر - السيراميك	معدن يخلط مع المعادن الاخري لاعطائها الصلابة والقوة وخصوصا النيكل والكروميوم وده اللي بيخلي المريض الواحد دايما حساس للتلاتة معا. Usese : Jewelry, dental plates, prostheses, polish stripper, hair dyes, ceramic, enamel صناعة الفخار والطوب.... NB :Patch test to it may produce specific reaction called Poral which is erythematous violaceous dots not due to cobalt allergy but due to allergens residing in acrosyringia.
Bacitracin	As neomycin. Cross reaction with neomycin may occur. NB: rarely cause anaphylaxis or contact urticaria.
Corticosteroids type I, IV hypersens	May cause ACD suspected by failure of response or worsening; may be ± d.t Cs itself or d.t preservatives 4 classes of Cs are known: A. Hydrocortisone and tixocortol group B. Triamcinolone acetinoide and budesonide group C. Betamethasone group D. Hydrocortisone 17 butyrate. Tixocortol and budesonide are best used as screaning to ACD to Cs.
Paraphenylinediamine	الحنة - الصبغات

Epoxy resin → dental filling	
	Other common allergens
Plant allergens	Primula, Poison Ivy, Tulip bulbs, Onion & Garlic. Poison ivy:(نبات يشبه اللولب) Is The classic example of acute ACD in North America. ACD from poison ivy is characterized by linear streaks of acute dermatitis that develop where plant parts have been in direct contact with the skin (مهم)
Plant photo-toxins	Parsnip, celery, parsley, fennel, orange. البرتقال
Chromate Commonest in men	Cement(مهم شفوي), leather, bleaches, matches, tattoos. NB1: it is of less problem in enlightened countries that insist to add ferrous sulphate to cement to produce water soluble chromate content. NB2: cement may cause ACD in hidden body areas through its dust or vapour.
Paraphenylenediamine	Coloring agent in: Hair dyes Henna (علشان يعطيها اللون الغامق) Textiles . خلي بالك كمان بتبقي موجودة في مادة حشو الاسنان فممكن تعمل: lichenoid drug eruption in mouth. NB: what are other complications of *henna?*.
Ethylenediamine	Preservative in creams, paints, rubber, antifreeze.
Topical medications	*Antibiotics:* Neomycin, quinoline, chloramphenicol, creams. *Antihistamines:* Antazoline. Anaesthetics: Benzocaine.
Colophony	Sticking plaster, solder flux, polish, varnishes.(الحساسية من البلاستر)
Parabens	Preservatives in cosmetics and creams.
Epoxy resins	Glues, surface coatings, PVC products.
Thiurams	Rubber, fungicides, hair dye, stockings, clothing dyes.
Rubber	Rubber dermatitis is due to accelerators, antioxidants and other chemicals used in manufacture of rubber rather than rubber itself. Sources are: car tyres, gloves, adhesive tapes and shoes. Rubber latex currently is the most important source of allergic contact urticaria (see Latex Allergy). The term hypoallergenic may refer to gloves that do not contain sensitizing chemicals added to rubber latex but may not indicate whether the gloves are rubber latex free. Some individuals may have delayed specific contact sensitivity to rubber latex, but contact urticaria to rubber latex is much more common than allergic contact dermatitis to latex.

C. Regional ACD

Q جه سؤال قبل كدة + واكتب عن Allergens كلها المشهورة

Region	Possible Cause of ACD
Scalp	relatively resistant to ACD. but it may occur d.t: → Ⓔ fragrance mix ◦ hair dyes ◦ Hair sprays ◦ shampoos ◦ Hair straighteners (الجلات) دايما مش بيحصل في الـ scalp ولكن على الأذن والوجه > scalp + postauricular & Neck
Face	- In general; CD of Face may be: 1- ACD → Face Creams, Powders, shaving lotions 2- Airborne ACD → ACD involving sun exposed & protected areas (Eyelid - postauricular & below chin) → البص عليهم الأول (حبوب اللقاح + dust of Cement) 3- Photoallergic CD → as ② but differentiated From it by sparing of sunprotected Areas. (مكان معرض للشمس) 4- Irritant CD → Subclinical ICD may be d.t Some Cosmetics. C/P < burning, stinging, or Contact urticaria (تبشوفها كتير في موسم الشتاء) DD ACD
Ear	- Rim & Postauricular → دايما حاجة اتحطت على الـ scalp وعملت ACD (الجلات..) - النضارة - Lobule → Nickle ACD (earrings / piercing) (حلقة / خزمة) - EA. Canal → 1- استعمال نقط للأذن للعلاج تحتوي على Thimirosal أو "Neomycin" or "bacitracin"

Eye lid (سبب)

- unilat → ACD
- Bilat → AD & Recurrent seb derm

1- Nail polish: Commonest site of Nail polish ACD is Eye-lid dermatitis (unilat).
2- Mascara
3- Fragrances.
4- False Eye Lash Adhesive.

Lips & Perioral

- Lips ACD:
  - Lipsticks
  - Tooth paste
  - Nail polish.
- Perioral ACD: ↓ Extension from Lip CD.
- Lips & peri-oral ↓ Citrus fruits & Mango → phototoxic Lip & perioral Dermatitis. (المواد)

Neck: (الرقبة)

1- Perfumes → ACD or photoxic CD.
2- Nickel → of Clasps of Necklaces (حبة السلسلة)
3- Textiles (نسيج) → Collar like dermatitis or "Neck side dermatitis"
4- Air borne ACD: so ACD in this case is sharply limited by Collars.

Trunk

→ uncommon site of ACD

- Axillary ACD may be d.t.
  - ① Axillary Vault → deodorant (Apex)
  - ② Axillary Folds → clothing dye.
- brassires may cause ACD from:-
  - Material itself
  - Elastic & metal (snaps) (مشبك)

Abdomen

1- Waist line: "Rubber dermatitis" from Elastic in pants & undergarments. (...بنطلون)
2- periumbilical: Nickel in Jeans Button or Stud.

Groin

1. Groin → usually spared.
2. buttocks & upper thighs: clothing dyes
3. Penis:
   - poison Ivy
   - Condom ✓
   - Transmitted by fingers (وفيه حالات ACD)
4. Vulvae: (50%) of pruritus vulvae is d.t ACD
   From: medicaments, Fragrances & preservatives
5. Perianal:
   - Medicatns: Caine, Neomycin
   - Cleansers: contain preservatives

Upper limb

- Wrist
  - Nickel: الساعة
  - Chromes leather wrist bands.
- Hands:
  - Site ± dorsum, Palms, Ring site, Web spaces, Finger tips
  - Allergens: Florists, Nickel, chrom, Rubber, Poison Ivy
  - Pattern: may be Pompholyx like, Nummular, streaks.

L.L

1. Shins: "Rubber" From Elastic stockings
2. Feet (shoe dermatitis):
   - Rubber
   - chrome tanned leather
   - dyes
   - Adhesives

drugs: Antibiotics & moisturizers applied to stasis D.

TABLE 2-2 LOCATION OF CONTACT DERMATITIS AND SUSPICIOUS AGENTS

Location	Suspicious Agent
Eyelids	Nail polish, eye makeup, airborne allergens
Earlobes or neck	Metal jewelry
Forehead, scalp margins	Hair dyes
Face	Cosmetic fragrances and preservatives, airborne allergens
Axilla	Deodorants
Hands	Gloves, occupational contacts
Waistband	Elastic
Dorsal feet	Shoes

• Invs. of ACD

Ⓐ. Patch Test
Ⓑ. photopatch test
Ⓒ. ROAT test
Ⓓ. Dimethyl-glyoxime test

Ⓔ Histopathology → see (Ecz.)

Ⓐ - Patch test "اختبار"

- def.
- Types
- Methodology
- Results.

½h
24-48h
7-21d

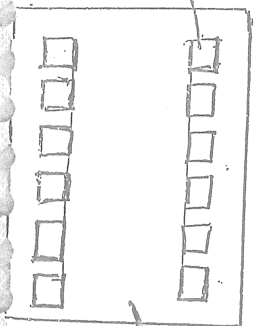
def.: special test used to detect Hypersensitivity to a specific substance (Allergen) that is in contact with the skin so that the allergen may be determined & Corrective measures taken.

Patch Cure CIP

Types

- **TRUE test** [standard screening Test] (Thin Layer Rapid use Epicutaneous test) جاهز
  - prepackaged Allergens
  - No of Allergens (23) (there is one control ⇐ accounts For the 29th Spot) → فارغين
    ↓
    (Ag 23) لأنه متسالين على فارغين أصلا عكس يطلعوا كلهم true لو عنده حساسية
- **Finn chambers test** (Individually prepared Aluminium chamber mounted on Scanpor Tape) جهز يدويا
  - Allergens come in multi-System Syringe & placed into Finn chambers mounted on Scanpor Tape
  - allows Testing For (50)+ Allergens (Expanded Series of Allergens > TRUE Test).

يعني باختصار



TRUE test:

- مربع بلاستر كبير كل واحد بيحتوي على ١٢ مربع صغير بيحطوا على الـ Allergens المراد الكشف عن الحساسية لهم
- وعلشان بيشترى جاهز فهو سهل الاستخدام
- ولكن عدد المواد (Allergens) الموجودة فيه صغير (٢٣)
(Prepacked Allergens)

Finn Chambers Test :-

- يتم تحضير الـ "Allergens" المراد اختبارها في سرنجات ثم يتم وضعها في حجرات من الالومنيوم (لانه خامل) مش بيعمل حساسية مثبتة على بلاستر وتوضع على الظهر (Scanpor tape).
- يتم اختبار عدد كبير من الـ "Allergens" أكتر من TRUE test
(Expanded Series of Allergens ≥ 50)

NACDG Series **or** European Standard Series

وغالبا كل بلاستر بيبقى الموجود عليه حوالي ١٢ حجرة تحتوي على ١٢ Allergen

So: if TURE Test is -ve
↓
Finn chamber test

(يعني لو الـ ٢٣ مادة طلعوا سلبيين نبحث عن مواد أخرى ايجابية في Finn test)

احفظه

. Methodology:-

(1.) Ask the patient To bring all his own materials For testing:-

يخلي المريض يجيب كل المواد اللي بيتعامل معاها في البيت أو الشغل علشان نعرف مكوناتها ونكشف عن الـ "Allergen" المسبب للحساسية

ابعت المحل قبلها بأسبوع

→ Bring or send all chemicals for Testing (1 W) before the 1st appointment

→ only small amounts required (Few drops or grains)

مش مهم الخطوة دي

. then these products are classified as:-

- leave on products as:
  - Moisturizers
  - Make up
  - ↓ Tested as it (من غير تخفيف).
- rinsed off products as:
  - soaps
  - shampoos
  - ↓ Tested after dilution.

وبعد ما نعرف المادة الفعالة في كل منتج بيتعامل معه المريض بنخففه بـ Petrolatum (بتركيز معين على حسب كل مادة)

وكده يبقى حضرنا المادة المراد اختبارها وتم وضعها في finn chambers

احتياطات قبل وبعد

(2.) Precatations:-

(قبل ما نلزق الـ Patch)
- Avoid back Exposure for sun (4 Ws) before Testing قبلها
- Avoid (Cs) at least 1-2 Ws before Testing (≥15 mg prednis.)

(بعد ما نلزق البلاستر)
- (No) Swimming, baths or EXcercise → ممكن البلاستر يتشال
- Arrange for someone to remark the test sites c̄ "indelible" Felt tip Marker

ارسم حوليه علامة علشان لو ما يتحركش من مكانه

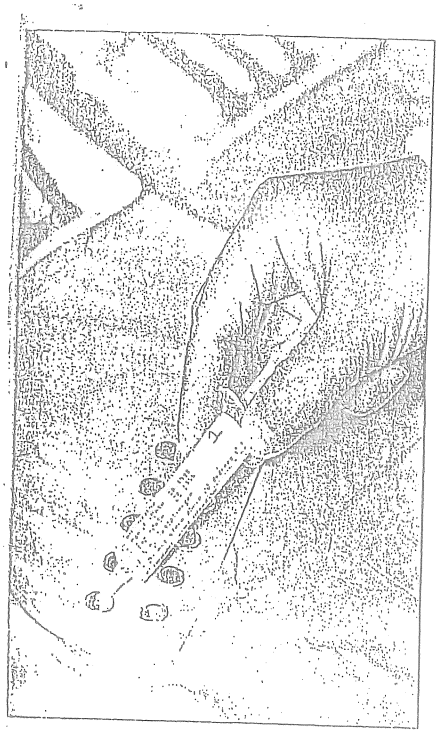


Fig. 15.11 Allergens contained within syringes being placed by nurse into Finn chambers.

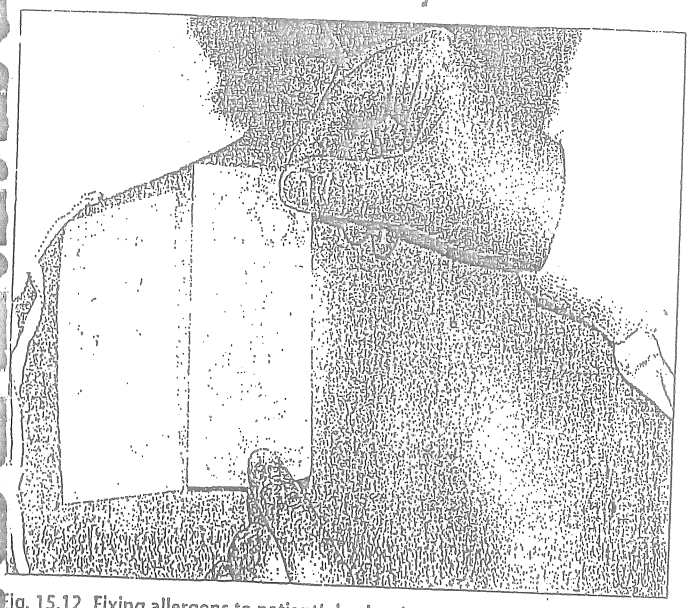


Fig. 15.12 Fixing allergens to patient's back using Scanpor® tape.

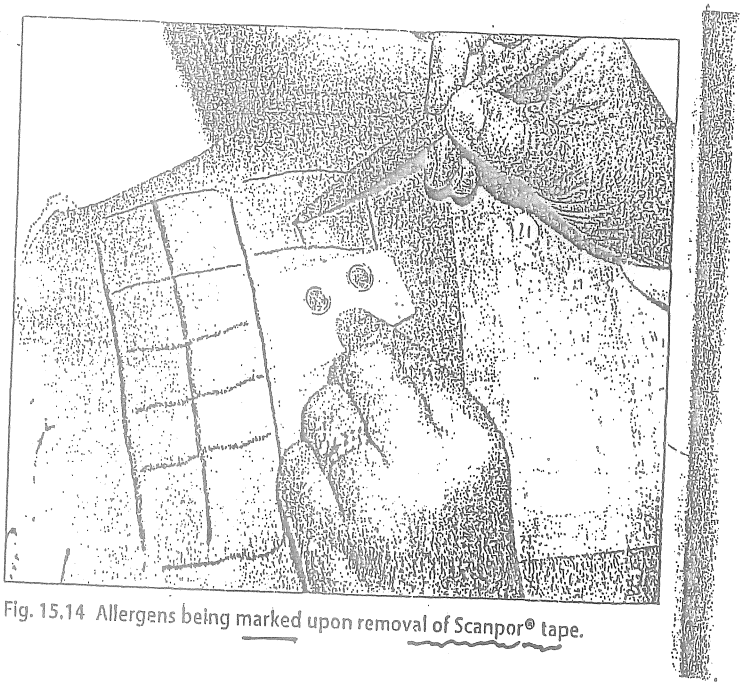


Fig. 15.14 Allergens being marked upon removal of Scanpor® tape.

اقرأ

3- Reading:

- The patient returns after 2ds (48 hrs)
وبيتأكد من ان البلاستر لسه لازق
كويس (هجرات Finn بتيجي عامله علامات
على الجلد)

بيأخد قراءتين:
- وقت الغاء] 2ds
- بعد الغاء في نهاية الاسبوع] 1w

① Patch is removed & Readings are taken:-

① Readings taken at:

1st reading → · 48 hrs after application (بعد الغاء)

2nd reading → · 3 - 7 ds (after application) Why

→ Some allergens as Gold & NeomYCin. dyes may be delayed.

النتيجة

· Results:-

(According to International Grading System for patch test)

- (−) → Negative react
- IR → Irritant reaction (← sweat rash, Follicular pustules, burn like react) ICD
- +/− (doubtful) → Faint macular Erythema. (equi-vocal)
- \+ (Weak +ve) → Non Vesicular reaction (only ← Erythema, papules, infilt)
- ++ (Strong +ve) → as (+) but there is Vesiculation
- +++ (Extremely +ve) → spreading bullae or ulcer.

الظهر كله غضبان

على Q . NB : Angry back Syndrome = Excited skin Synd.

def.: state of Hyper excitability ch^by False +Ve reactions (the -Ve Tests appears Weakly +Ve). it may be d.t:

AET: (1) Active dermatitis at time of patch testing or

(2) strong +Ve reaction → induce local skin hyperreactivity in areas where patches are applied.

hypoallergenic Plaster → البلاستر نفسه

نوع واحد وعل ظهر كله

Managment: . re testing the patient Sequentially to Small series of these Allergens. أخفف

لو كان السبب رقم ② / لو كان السبب رقم ① → ↓ # of Active Dermatitis.

. B- photopatch test = (for ⊠ of ...)

. This test used for evaluation of photoallergic CD

To such Substances as:
- Sulfonamides
- Phenothiazines
- PABA → in Sun Blocks
- Oxybenzone → in Sun Blocks

على ظهره : these : Chemicals are applied in duplicate Sets.

. one set : receives 10 J/cm² (or 1 J/cm² less than MED to UVA).

. other set: the other series protected From UVA

(i). +Ve React^ on Both sides → ACD

(ii). +Ve " at UVA exposed side only → PACD

C-ROAT Test

اجربه على ايدى مثلاً

(Repeat Open Application Test)

Indications: (1) -ve patch test despite there is strong suspicion of ACD.

(2) Weak +ve patch test (specially for leave-on consumer products)

Ref. Secrets (remember that: patch test is one time occlusive test that doesn't duplicate low level chr. daily Exposure)

← هاتجيب المادة دى (بالجودة مثلاً فى مطبخ) وتيجى استعمالها مرتين يومين على جانب الرقبة - خلف الأذن - أو على الذراع لمساحة صغيرة كده

لو حصل ← ACD ← "the weak patch test is highly Revelant"

D. Dimethylgloxime test

def useful & practical way to Identify Metallic objects that Contain enough Nickel to provoke ACD to Nickel.

- عكس دكتور الجلدية يعمله فى العيادة أو المريض يشترى الـ Kits ويعمله فى البيت.
- توضع نقطة بسيطة من هذه المادة على وطية أو شارشه ويتم تدليكها بالأشياء المواد الكاشفة عن النيكل فيحدث فى خلال ثوانى لو اتحول لونه لشارش إلى أحمر ← +Ve For Nickel-

---

- Treatment of ACD
  - ① Remove the Allergen ××
  - ② Treat the Eczema كما يجب

Irritant CD (ICD)

def. Cutaneous inflammatory disorder resulting from direct cytotoxic effect of chemical or physical Agent (Non Immunologically mediated).

Epidemiology :-

- represent 80% of cases of CD
- represent 80% of occupational skin disorders ← ميكانيكي، تهيج، إصابة

Pathogenesis : → 3 main pathophysiological changes :

Cytotoxic + 2D (disrupt, denat.)

① Skin barrier disruption → ↑TEWL

② Epidermal cellular changes. e.g Keratin denaturat^n.

③ Cytotoxicity: ↓ release of ← TNFα, IL6, IL1B → cytotoxicity

- ICD has 2 phases: (HL)

Acute

⊙ Penetrat^n by the irritant Agent produces 3 changes (إلتهاب)

- The Most important cytokines are:-
  - TNFα
  - IL6
  - IL1B

  → upregulate Expression of ICAM-1

  ولا IL8

chronic

أهم مرحلة هنا هي damage to Stratum Corneum Lipids (و mediate St. Corneum barrier function)

① loss of KCs cohesion → desquamat^n

② ↑TEWL

Causes of ICD:-

- Almost (any) material can cause ICD if there is sufficient exposure both in ① Time & Concentration ②.

→ **Most Common irritants are:**

IRRITANTS AND MECHANISMS OF TOXICITY

Irritant	Mechanisms of toxicity
Detergents	Solubilization and/or disruption of barrier lipids and natural moisturizing factors in the stratum corneum Protein denaturation Membrane toxicity
Acids	Protein denaturation Cytotoxicity
Alkalis	Barrier lipid denaturation Cytotoxicity through cellular swelling
Oils	Disorganization of barrier lipids
Organic solvents	Solubilization of membrane lipids Membrane toxicity
Oxidants	Cytotoxicity
Reducing agents	Keratolysis
Water	If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells

Table 16.2 Irritants and mechanisms of toxicity.

(Margin notes: Detergents – منظفات و مطهرات; Acids – Hydrofluoric & sulfuric acid; Alkalis – NaOH, في منظفات صابون وأملاح تجميل; Organic solvents – بنزين و جازولين)

- Acids
- Alkalies
- Detergents
- Disinfectants
- Solvents
- Plastics,
- fabrics
- Water
- Bodily fluids: ← Saline, urine, feces.
  - diaper D.
  - lip licking
  - Fescarnet ...
  → Periurethral ICD
- Food & its additives

Q (Cement) • ملاحظة: الأسمنت ممكن يعمل تشوهات

① **ACD**: d.t chromate

② **ICD**: d.t its Alkalinity

[2 Types]:

- **Acute delayed** – بيحصل بعد (٨ – ٢٤) ساعة
- **Cumulative** (تراكمي) – بيحصل بعد (شهور – سنين) من التعرض المتواصل → المريض بيشتغل طول عمره في الأسمنت والأملاح (ك عامل بناء)

3. **Both**: ACD & ICD (ACD may accompany [Cumulative] ICD)

· Clp of ICD

As General picture of Dermatitis (or) Eczema : it may be :

- Eczematous
- Non Eczematous
  - Burning sensation
  - Chemical Burn like
  - Pustular ✓
  - Acneiform ✓
  - folliculitis ✓
  - Keratic ✓

- Acute →
- Subacute : →
- Chronic : →

Types of ICD : → 3 (Subclinical, Subjective 3)

- Acute Immediate
- Acute delayed
- Chr. Cumulative

- Irritant Reaction ICD
- pustular & Acneiform

- non Erythematous (subclinical)
- Subjective or Sens.
- Asymptomatic

- Traumatic
- frictional
- Air borne

1- Acute Immediate ICD (minutes-Hours)

strong irritant
short period

2- Acute delayed ICD : (8 - 24 hrs)

e.g anthralin

3- Cumulative ICD : (chr. ICD)

weak irritants
Repeated Application

occurs as a consequence of ① multiple subthreshold skin insults ② without sufficient time bet. them for complete barrier function repair.

→ Cumulative ICD ↓ develops (e) subsequent exposures (or) when i threshold of irritants is ↓↓ e.g AD. (then)

(4) Pustular or Acneiform [folliculitis] e.g mineral oils, tar

(5) Non Erythematous ICD: Pathological ICD (Not) clinical (sub-clinical)

(6) Subjective (Sensory): → only stinging / burning sensation

(7) Air-borne ICD: تيار الهواء

(8) Frictional ICD: d.t repeated frictional Trauma (hyperkeratotic, Acanthotic, lichenified)

(9) Traumatic ICD: Irritant (is) applied over sites of Trauma e.g Burns & Lacerat

(10) Asteatotic ICD: [see Xerotic Ecz.] ✓

(11) irritant reaction ICD: subclinical (dz) wet chemical environment (hair dresser)

Sharp outline — extend beyond site

TABLE 2-1 COMPARISON OF IRRITANT AND ALLERGIC CONTACT DERMATITIS

	Irritant (80%)	Allergic (20%)
Examples	Water, soap	Nickel, fragrance, hair dye
Agents: Number of compounds	Many	Fewer
Distribution of reaction	Localized	May spread beyond area of maximal contact and become generalized
No Conc.: Concentration of agent needed to elicit reaction	High	Can be minute
Time course	Immediate to late	Sensitization in 2 weeks; elicitation takes 24–72 h.
Mechanism: Immunology	Nonspecific	Specific type IV delayed hypersensitivity reaction
Diagnostic test	None	Patch test +
IL 4		
Incid:	More Common	less Common
clp:	stinging or Burning	Itching
prognoses	Better	Bad → Painful

Incid, Mech, Agents — clp, Diagnosis

• Diagnosis of ICD: No specific test, Diag
• of exclusion; when Dermatitis can't be explain
by -ve Patch test to a known allergen.

Photo Contact Dermatitis.

DNNZ.

def. Allergic or Toxic reactions occurs When certain chemicals are applied to the skin & subsequently Exposed to sun.

لازم وجود العامل ده

(Topical Agent + UVR ⟶ photoContact dermatitis)
(photosensitizers)

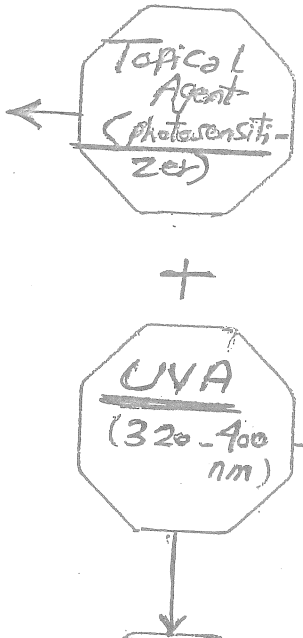
- Pathogenesis : see drug induced photosensitivity (Exogenous photoreactions or dermatoses).

- Examples :-

1- Sunscreens :-
  - Salicylates
  - PABA
  - Benzophenones
  - Musk

2 fragrances :
  - Musk

3 others :-
  - 5Fu
  - Tar
  - Psoralens
  - sulfa.
  - Cadmium



لو دهنا فاتجاش

→ also UVB & Visible Light (any light length 280-600 nm)

Photocontact D:
photoAll. غالبا توجد Photosensitizers
- Topical → Photoallergic CD
- Systemic → Photoallergic & Phototoxic

سابق للتفرقة بين النوعين see light & skin.

للمقارنة

see photoreact.

- Photo. ACD → Eczematous Eruption (No Post-Hyperpigm).
- Photo. ICD (Phototoxic CD) → sunburn like Erupt. (with Post PIH Hyperpigm)

(on areas Where the Drug applied & UVR Exposure).

NB Examples of phototoxic dermatitis:-

(1-) Phyto photo dermatitis:

- Contact ē plants containing phototoxic substances + UVR → Reactⁿ
- → clp: streaky or bizare pattern
- → the most common plants are those of Umbelliferae family (Paisnip, Celery, Parsdy, Hogweed)

Better classified as Phytophoto D. rather Photo D.

(2) Berloque (Berlock) Dermatitis:-

(Berlock = Trinket or charm)

def: phototoxic reaction d/t Contact with plants contg Oil of Bergamot + UVA Exposure.

- the photoactive Component is:- Bergapten (or 5 MOP)
- Source:
  - Bergamot lime plant
  - Perfumes
  - Fragrances
  - Cosmetics
  - Toiletries
  - after shave lot.
  - Colognes
  - Sunscreens
  - Moisturizers.
  - Soap . Detergents.
  - air Freshness.

Oil of Bergamot (80-120 mins Contact to skin) ← Mid-late summer

+

UVA

↓

Berloque Dermatitis

CIP → 2 phases: → Acute inflamm.
→ Hyperpigm.

1- Initial acute inflammatory phase: after 24hrs

- Erythema
- Edema
- Vesiculat⁻
- Pain (not pruritic)

→ bizarre config.

Pattern of lesions: Pendant-like or drop like

2 2nd phase: after 1-2ws. → Hyperpigmentat⁻ (chief Complain

- site: any site where perfume is applied & UVA Exposure. e.g ♀ → sides of Neck, arms, Trunk.

NB:- usually → (No) acute inflammatory phase & the presentat⁻ is only dt Hyperpigm.

- durat⁻ of: Acute phase → ds- ws

Hyperpigm " → ms- ys.

- Invs ① photopatch test (see photosensitivity)
② Pathology (H/P)

- Treatment:-

1- Avoid all preparations containing oil of Bergamot

Emollient / Bleaching cr

2. Acute phase → wet Compresses (Antiseptic lot) ± Analgesics + Sunscre

3. Hyperpigmentat⁻ (self limiting but after long period)

- Sunscreen
- Hydroquinone 2% (1x2)
- Kligman's formula (??) ~ بعمل PIH

. Hydraquine
. Tretinoin
. Dexamethasone
. Ethanol
. Propylene glycol

→ - Ellagic acid: Naturally Existing polyphenol That -- Tyrosinase by Copper chelat⁻

تابع الـ

Exogenous Eczema

- Dermatophytide → see fungal inf.
- Eczematous pMLE → see pMLE.

Infective Eczema
(infective eczematoid dermatitis, microbial eczema) → « Caused by infection »

- def: Ecz. Caused by contact with Microorganisms or their products & cleared by eradicat° of the organism.
- C/P: - Seen few cms from 1ry site of inf.
  - Can be seen in:-

Mechanism
Bact. Ag
↓
Cytotoxic reactn
↓
Ecz.

1- Flexures (in scabes)
2- Scalp & post auricular areas (in Pediculosis & impetigo).
3- around discharging Sinuses, Wounds & ulcers.
4- Around Molluscum lesions (حوالين الجدري) [Molluscum dermatitis]
[5- Complicating T. pedis (d/t G-Ve bact.)
6- Microbial feet Ecz: Weeping & Crusted Ecz. seen on the dorsoMedial aspects of Toes in persons ē bad Hygiene or T. pedis.

ماهو الفرق بين الـ Infected Ecz & Infective Ecz. → ecz ē super infection over it
← في فرق طبعا ولكن تفريقهم كلينيكلي صعب

7- infective dermatitis in children ē HTLV-1:
- Severe Type of Ecz. Seen in Jamican children in ass. with HTLV-1 usally Generalized & Severe (Scalp, Postauric, Eyelid, Nose perianal).
- may be an important marker for HTLV-1.

- Tt → Systemic antibiotics Preferred as Topical ones may → CD.

syst AB

Atopic Dermatitis (AD)

- def of Atopy: a = No or without
  Topy = Topos = place → out of place = غريب
  strange = unusuall.

معناها → Atopy is an inherited tendency to develop استعداد
a hypersensitivity response (Allergy) to certain Enviromental stimuli (Allergens) in presence of chic Immunological profile.

- Atopic dermatis (AD):-

→ Cut. manifestations of atopic diathesis, استعداد - تهيئ للإصابة
Ch By chr. relapsing, intensty pruritic. dermatitis ē chic distribution pattern In Atopic individuals.

* Atopic disorders or diathesises are:-

(5)
- Asthma.
- AD
- Hay fever (Allergic Rhinitis)
- Allergic Conjunctivitis
- Allergic Gastroenteritis.

" Etiopathogenesis "

1. Genetic factors
2. Immunological Factors.
3. External factors = trigers
4. others
   - Hygiene theory
   - Abnormalities of sweating
   - Change in cutaneous lipids
   - ↓↓ pruritus threshold
   - Change in PDE activity
   - Altered vascular and neurocutaneous reactivity VC

Autosomal جسمي
p short arm
q long "

(I) Genetic theory (factor) in AD:-

- Autosomal dominant Inheritance (AD) ✓
- If [2 parents] are affected by AD: → (80%) Incid. of Sibling Affect
- If [1 parent] is " by AD: → (60%) Incid

Xerosis ← عشان كده غالبا → • Deffective filaggrin Gene (20-50%) (FGL) on [19/12.3] → (1q21 chromosome long arm segment)
± Ass ē Ichthyosis vulgaris.

Defective barrier → [↑ TEWL] (Trans epid water loss)

II. other Factors

(i) [Hygiene theory]: ↑ Exposure to Infectious Agents during early life → (++ Th_1) to be potent >> Th_2 → ↓ Incid. of AD.
← يعني الواد مايكروبس بيقا ابن البيئة أو البلد النظيف عيان —

(ii) [Abnormalities of Sweating]: in Atopics → ↑ sensitivity of Glands to Catecholamines → ↑ Sweat → taken up by st. Corneum → gland obst. → Sweat Retentⁿ → gland Rupture → diffuse to dermis → Itching.
العرق ↑

(iii) Abnormalities in cut. lipids:
cut lipid
- ↓ Glandular, Ceramide & Linoleinic
- ↑ Epid. lipids.

} → All cause Xerosis.

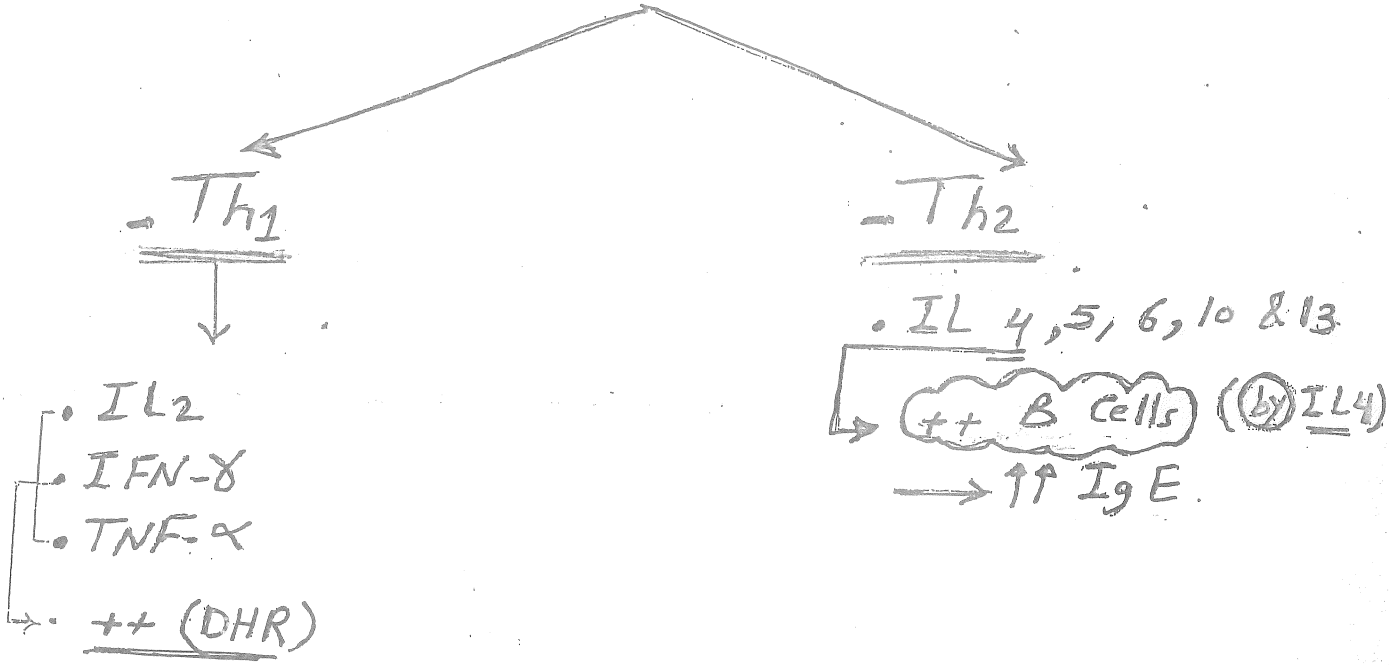
(IV) ↓ pruritus threshold: Early & prolonged experience of itching
الأرش

(V) Altered Vascular & Neuro cut. Reactivity:-
VC
"Paradoxical VC"
↓
ثانوي
So Atopics have Pallid skin & White Dermatographism.

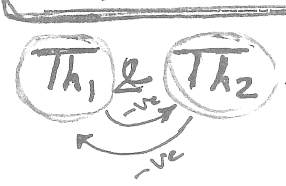
(II) Immunelogical factors:
- T-cells
- Igs
- LCs
- Eosinophils

T-Lymphocytes

- CD4 or T-helper cells are of 2 subtypes:-



- In NL individuals: there is balance between Th1 & Th2 → each exert inhibitory action to the other



- In Atopics: → Th2 predominates in Acute & subacute phases while Th1 predominates in chronic phase.

So:

The Immunological Profile in Patients of AD is (eg/acb) Biphasic :-

Th2 predominate in acute & Subacute lesions (Early phases)

1. ↑ IL 4, 5, 6, 10, 13
2. ↑↑ IgE
3. ↓ IL2, TNF-α, IFN-γ
4. -- Type IV Hyper-sensitivity Reactn (↓ Th1)

↓ Eliciting allergic (ACD)

↑ susceptability for Fungal & Viral inf.

Th1 predominate in chr. lesions (Late phase)

- ↑ IFN-γ, IL2, TNFα
- Reversal of Type IV (delayed hypersensitivity reactn suppression).

IL4 level correlate ē dis. activity

NB: ↑ IgE level → ↑ susceptibility to IgE Mediated diseases: e.g Anaphylaxis, urticaria, Atopy.

Immunoglobulins

1. ↑ IgE production.
2. ↑ IgG1 (ass. ē air born allergen Hypersens)
3. ↑ IgG4 (ass ē Food Hypersensitivity).

NB → ↑ IgE → not always a Constant finding (See intrinsic AD)

LCS (Langerhans cells) →
- ↑ No
- ↑ Reactivity
- ↑ Expression of "FcεRI" of IgE.

Eosinophils :→ Peripheral Eosinophilia / Hyperreleasable in response to non Allergens e.g Wool

↑↑

(III) Extrinsic factors

(1) Irritants & Contacts: ← Solvents, disinfectants, Latex, Nickel

(2) Airborne Allergens: ← House dust mites, Pollens, moulds, Mites.

(3) Food (Diet): ← Milk, Soya products, Eggs, Nuts

لبن، هوبا، بيض، البندق واللوز

علاقة الأكل بـ AD تتلخص في الآتي ؟؟

① تؤثر على مجموعة صغيرة من المرضى وخصوصا الحالات الشديدة.

② الأشخاص الذين ثبت حساسيتهم من الأكل هم الذين يحتاجون يتفيدوا من وقف هذه الأصناف.

③ الحافظ على لبن الأم وتأخير اللبن الصناعي [تأثير ضعيف ومؤقت على السنة الأولى فقط].

→ **So food plays a minor Role**

(4) Microorganisms: (2) Common organisms may act as a supar antigens → Exacerbate of AD:

- Staph. aureus
- Malassezia

Severe AD of Face → Lotriderm or Pivasone cr

↳ in head & Neck Dermatitis IgE to M. Furfur can be demonstrated → Good Response to Antimycotic

(5) Hormones ← Preg. menopause Menses] (all) Exacerbate AD.

(6) Stress : ↑ AD

(7) climates ← Winter: ↑↑ (عكس الصيف) Summer: ↓↓ (عكس الشتاء)

Types of AD (Extrinsic & Intrinsic)

	Intrinsic (Non Allergic)	Extrinsic (Allergic)
• Incid.	Less Common (20%)	Common (80%)
• Age	Late (Adulthood)	Early (childhood)
• FH	Less +Ve	• strong (+Ve)
• Ass. other Atopic Manifs. (diathesis)	Rare	• Common ↑
• Mechanism:	Non Immune Mediated.	• Immune Mediated. (Type I)
• Triggers	• (No) ← Food Aeroallergens • Irritants	• food, AeroAllergens & Irritants.
• IgE	NL	↑↑
• Cytokines: - IL 4 & 13 - IL5 & IFNγ	↓↓ No difference ↑↑	↑↑ - No difference.

• Intrinsic: (NO)
(⇐ Ichthyosis
⇐ Hyperlinearity
⇐ FLG → NL barrier

• Epidemiology: Incid. AD affect (15-20%) of children & (1-2%) of adults.
• Age: (85%) of cases appear in 1st year & (95%) appear ≤ 5 Ys.
• Sex: M:(F) = 1:1.4

• CIP → 4 phases:
1. Infantile (2ms - 2Ys)
2. childhood (3Y - 11 Ys)
3. Adolescent / Young adult (12 - 20)
4. Adulthood (> 20Ys)

C/P [Age distribute
Morphology
clearing]

(4 phases) →

Acute Weeping Ecz. → Lichenif. ± Weeping → Lichenif → Lichenifict. fissures ± vesicles

Atopic dermatitis may present at any age, but 85% of patients experience their first outbreak by their first birthday, and 95% by their fifth. Four clinical phases are recognized:

1. Infantile (2 months–2 years)
   - Distribution: Cheeks (Fig. 8-1A), face and scalp, extensor surfaces of extremities (perhaps from crawling), and trunk
   - Morphology: Erythema, papules, vesicles/oozing, and crusting
   - Clearing: Dermatitis clears in half of the patients by 3 years of age ← 50%
2. Childhood (3–11 years)
   - Distribution: wrists, ankles/backs of the thighs, buttocks/ and antecubital and popliteal fossae (Fig. 8-1B) → خلف الركبة
   - Morphology: Chronic, lichenified scaly patches and plaques that may have crusting & oozing (see Fig. 8-1B)
   - Clearing: Two thirds of patients clear by age 6 ← 60%
3. Adolescent/young adult (12–20 years)
   - Distribution: Face, neck, arms, back, and flexures (Fig. 8-1C)
   - Morphology: Thick, dry, lichenified plaques without weeping, crusting, or oozing XX
   - Clearing: 90% or patients clear by age 18 ← 90%
4. Adult (>20 years)
   - Distribution: Most commonly involves the hands, sometimes the face and neck, and rarely diffuse areas
   - Morphology: Lichenified plaques, fissures on the hands, occasional vesicular outbreaks, one subset of "sensitive skin" patients

- Infantile stage:

XX - Diaper Area usually spared

- Exacerbat^n may occurs d.t:
  - Emotional stress
  - Teething
  - RTI

→ ⊙ Hill's Atopic Erythroderma, progression of condition to affect the whole body.

NB
- Dermatitis
  - < 3 ms → SD
  - But > 3 ms → AD

- Childhood phase:-
  - Fossae < Antecubital / Anti'popliteal
  - this Ecz is a mixture of:
    - Prurigo of Hebra → Extensor aff.
    - " " Besnier → Flexor aff.

- Adulthood Phase:
  - Persistance > 30 Ys → usually in Psychotic pt.
  - disseminat^n may occur → disseminated ND.

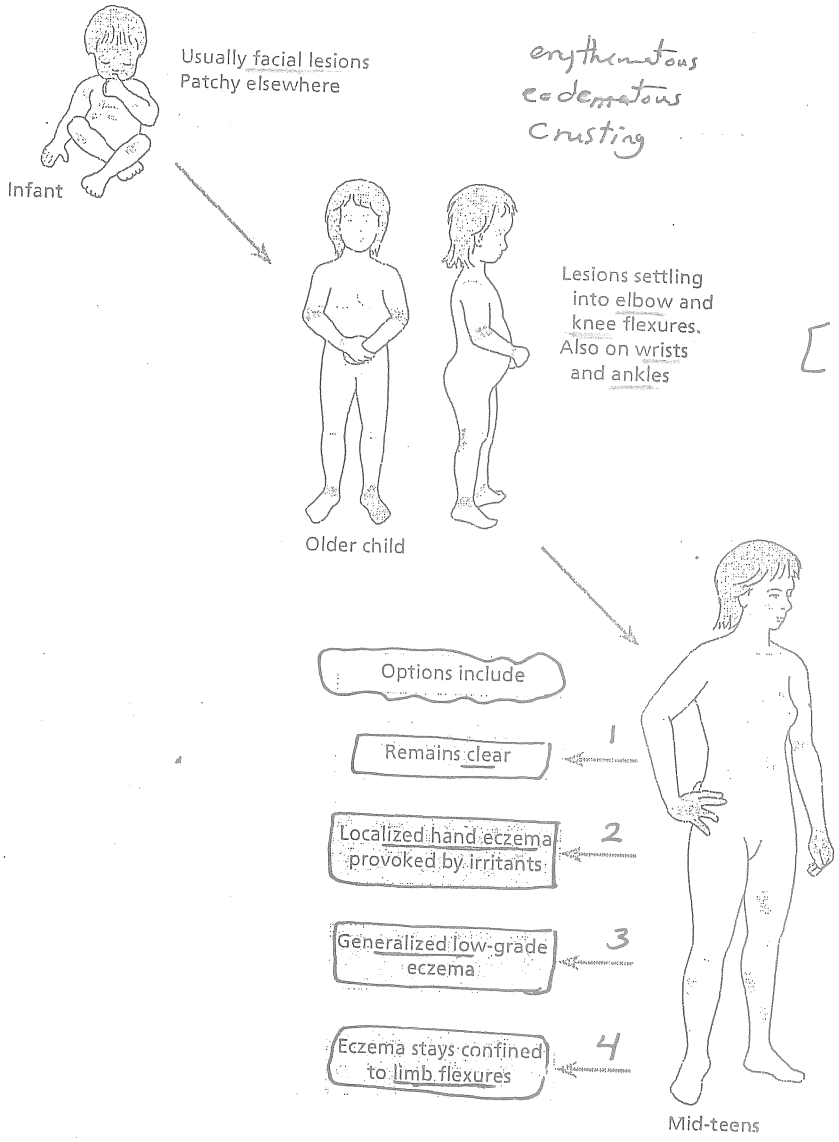


Fig. 7.12 The pattern of atopic eczema varies with age. It may clear at any stage.

Atopic erythroderma :-

1st few days of life

adults

(Un Common)

Diagnostic criteria for AD.

2 tables ① Hanifin & Rajka (1980) → the most accepted & widely used.
② American Academy of Dermatology Criteria (2003)
③ Williams criteria / UK working party (2006)

(A) Hanafin & Rajka

هانفين راجيكا

(A) Major criteria (4)

Cardinal Symptom

1- Pruritus
2- Typical Morphology & distribut^n < infancy: Facial & extensor involvement / Adults: Flexure lichenification
3- chronicity or chr. relapsing course
4- Personal or family history of Atopy. (+ve) FH

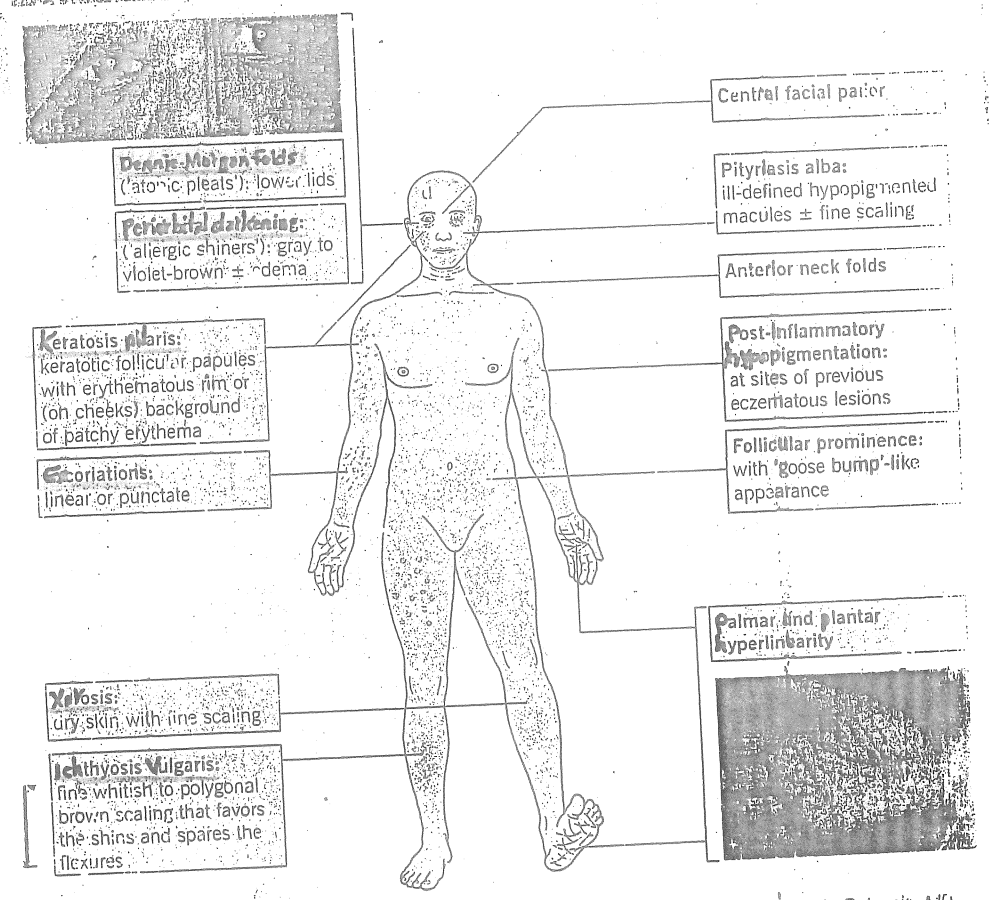


Fig. 10.10 Associated features of atopic dermatitis. *inset of hand: Courtesy, Jean L. Bolognia, MD.*

(B) Minor criteria "23"

- onset: Early (< 2m)
- Course: Influenced by < Environmental & Emotional factors.

- Intolerance to Wool & Lipid Solvents.
- IgE changes (↑level & Reactivity)
- Associated features: "3"

for D = 3 Major + 3 Minor

Associated features

1- Cutaneous:
- Cut.
- Eye
- stigmata.

- Xerosis
- Ichthyosis (↑ TEWL) due to FLG gene
- pit. Alba
- Keratosis pilaris. (KP = 40%)

- Reticulate Pigmentation of Neck (dirty Neck)
- periorbital milia

- White Dermatographism
- Cholinergic urticaria ✓

- ↑ susceptability to Inf ← staph. aureus. "Impetigo"
  HSV (Eczema Herpeticum)
  - Hand Eczema
  - Nipple "

2- Eye:
- Facial Erythema / paller
- Periorbital Accentuation

- Cataract.
- Keratoconus.
- Atopic Conjunctivitis.

3- Atopic stigmata: → are permanent (always)

- Eye ← periorbital Hyperpigm. (Allergic shiners) (always)
  Dennie-Morgan infra orbital folds (pleats)
  thinning of outer Eye brow (Hertoghe's Sign)
- Nose → Perinasal paller (Head light sign)
  → ↑ Nasal Creases.
- Hair → low set hair line. (تحت الحد)
- Neck → Ant folds.
- palms → ↑ creases. (Hyperlinearity).

B. American Academy of Dermatology (2003)

I. Essential features (must be present) must

1. Pruritus
2. Eczema *with:*
   - Typical morphology, distribution and age specific pattern :
     - a. scalp in infants → cheeks
     - b. facial, neck, and extensor involvement in children
     - c. current or prior flexural lesions in any age group
     - d. sparing of groin and axillary regions
   - Chronicity or chronic relapsing course.

II. Important features *(seen in most cases, adding support to the diagnosis)* most

1- Personal or FH of Atopy.
2- Early onset < 2ms.
3. IgE reactivity.
4. Xerosis

III. Associated features *(nonspecific clinical associations that help in the diagnosis of AD)* → all ass. features except (See Ass. Features)

1. Atypical vascular responses (eg, facial pallor, white dermographism, delayed blanch response)
2. Keratosis pilaris/hyperlinear palms/ichthyosis
3. Ocular/periorbital changes
4. Other regional findings (eg, perioral changes/ periauricular lesions)
5. Perifollicular accentuation/lichenification/ prurigo lesions

× - Xerosis
✓ - Add Vascular changes

IV Exclusion criteria: presence of other conditions of Itchy Skin e.g CD & Scabies ✓

*UK working party criteria (Wiliams criteria) (HL)

- Patients must have an itchy skin condition (or parental report of scratching or rubbing in children)
- Patients also must have 3 or more of the following criteria:
  1- History of flexural involvement, e.g. antecubital area, popliteal fossa, fronts of the ankles, or neck
  2- Personal history of asthma or hay fever or a history of atopic disease in a first- degree relative in patients <4 y of age
  3- History of generally dry skin in the last year
  4- Visible flexural dermatitis or dermatitis involving the cheeks or forehead and outer limbs in children <4y of age <2y.
  5- Onset younger than age 2 years (not used if child is <4 y)

NBs:

1- Other Presentations of AD
- Nummular dermatitis
- Eyelid dermatitis
- Ear dermatitis; post-auricular fissures
- Cheilitis
- Nipple dermatitis
- Facial dermatitis
- Hand dermatitis "F&"
- Juvenile plantar dermatosis + (Sole)

Dermatitis of:-
- Face
- Eyelid
- Ear, postauric
- Lip
- Nipple
- Hand . Sole
- Nummular

2- AD Associated diseases & Syndromes:- (Nigel)

A. Ass. diseases:-
- other atopic diathesises (30%)
- Eye → Cataract & Keratoconus.
- Ear → OM
- Skin → Ichthyosis . Vitiligo . AA . geographic tongue.

B. Ass. Syndromes:

Ataxia Telangiectasia (Ataxia + Telangiect. + sinopulm. inf + Leuk. ↓lymph)
Anhidrotic Ectodermal dysplasia (Anhid. + Alopecia + dental defect).
AEP (Dermatitis + Diarrhoea + Alopecia). — acrodermatitis enteropathica
PK (↓pigm. of skin/Hair + Deafness) = pigmentary dilution + Atopy — phenyl Ketonuria
Netherton synd. (Ichthyosis + AD + Trich. invaginata & FTT).
Comens synd. (FTT-Ens.↑, ↑IgE, ↓Igs + CuF < Eosin./Atyp. + Syst. L-N, HSM, diarrhea)
Hartnup dis. ↓ plagra + Neuropsychiatric + AA ↓ ↓ urine aminoaciduria
Histocytosis X. (niacin) - Wischott Aldriche purpura & eczema & thrombocytopenia
- Job Synd. & Hypereosinophilic Synd.

3. In atopic dermatitis, which comes first—the itch or the rash?
No primary skin lesion has ever been established in atopic dermatitis. Hanifin and Rajka suspect that all the cutaneous changes of atopic dermatitis may be due to friction and itch-induced scratching. As Beltrani states, "Atopic dermatitis is an itch, which, when scratched, erupts." If there is no friction or scratching, there is no eruption.

Itch —which cause→ Rash

what is the 1ry lesion of AD??

4. SCORAD Index = Scoring AD :-

A- Extent criteria

B- Intensity ll :

- Erythema
- Edema
- oozing
- Excoriation
- Lichenifications

→ كل واحد على حسب درجته Score تعطى له من 1-3

C- Subjective Symptoms :

- pruritus
- insomnia

Total score = $A/5 + 7B/2 + C$

Hyper-IgE syndrome (Job's syndrome) occurs in infants and children. It resembles atopic dermatitis but involves particularly the scalp, axillae and groins. The associated features include persistent secondary bacterial infection, fluctuant cold abscesses, contact urticaria, bronchitis and severe lung damage. The key laboratory finding is the great elevation of serum IgE (greater than 2000i.u / mL) often with some eosinophilia. Neutrophil chemotaxis is impaired.

The hyper-eosinophilic syndrome is a rare disorder, occurring in middle aged males, and characterized by intense blood eosinophilia. There is affection of internal organs (heart, nervous system. Liver, lung and gut). The skin changes include an eruption resembling atopic dermatitis or urticaria, itchy maculopapular eruption or erythroderma. Treatment includes systemic steroids and cytotoxic drugs such as hydroxyurea.

- AD, AR or sporadic
- Mutate in → STAT3 & Tyns. K2

النمط الوراثي

- 3 Lab ← IgE ↑ >2000 IU, ↑ Neutrophils, Eosinophilia

⑥ AD with Bad Prognosis:-

- Young age
- ♀
- (+ve) FH
- other Manifs. of AD (diathesises)
- Persistent Xerosis in old age.

- Complications of AD:-

1- Impetigmisation & 2ry bact. inf.

2- Folliculitis

Q سؤال امتحان → 3- Eczema Herpeticum (Kaposi varicelliform Eruption)

4- Ass. diseases & synds.

5- GR: in Severe Cases (>50%) of BSA.

- *Investigations*

A – Skin prick test

- Total IgE (RIST)
- Specific IgE (RAST)

-- Eosinophilia; blood, lesions

- Th2 cytokine profile; IL-4, IL-5, IL-13
- Reduced Th1 response; interferon-γ, DTH to contact allergens (INF-γ)

NBs

(1) Dennie Morgan folds:-

- Is d.t Eyelid Edema & itching → Existence of 2nd or 3rd Palpebral Crease.
- +Ve in → (75%) of Atopics, (5%) of Normals → So of Little Value in Diagnosis

(2) Some authors Considered infraauricular Folds & Fissures are diagnostic because they are (+ve) in 22% of Atopics, 3.3% of NLs.

Management of AD

(No Curative ttt)

1- General Measures :

- Bathing
- Clothes
- Exercise
- Environment /irritants & Contacts – exclusive breastfeeding 4-6m
- Diet (Probiotics – prebiotics) – low allergen diet for mother during breastfeeding.
- Health Care Education

2- Topical ttt :-

- Cs
- Cs + Antibiotics
- Calcineurin inhibitors
- Emollients
- others → Antipruritic
  - lox di Na Cromoglycate

3- systemic ttt :

- Antihistamines
- Antibiotics
- Cs
- tacrolimus
- interferon
- cyclosporine
- tomycine
- Zafirlukast
- Anti PDE
- IVIG
- others → MTX
  - Azathioprine
  - Mycophenolate

4- phototherapy

5- others therapies

17

A- General Measures

(1) Bathing

① الماء : يفضل ع البانيو ١٠-١٥ دقيقة

. استخدام ماء فاتر (tepid) [تجنب استخدام الساخن والبارد]

(جلده يكرمش) = hydration of st. corneum

② الصابون : • Should be:

① Non irritant e.g Dove, Keri, Neutrogena

② acidic soap (groin, Axilla) → staph ذى ستافيل

③ Antiseptics may be added to water الافضل as chlorhexidine. or كلوروكس ابيض < Na Hypochlorite 6% ½ cup for Full Tub or ¼ cup for ½ Tub. (5 mL → 4L)

④ Soap used at / Specifically at < Grion, axillae, anogenital, scalp. ??

إضافه حاجتين لماء البانيو
- Cyteal lotion
- فنجان emulsifying oil

③ اضافة مرطبات لماء البانيو : (emollients)

- as mineral or vegetable oils زيت زيتون
- Bleach bath

④ الليفة : (تكون اسفنج)

XX . Avoid < Rough ones / Vigrous Rubbing.

⑤ بعد الحمام: ١- يفضل ترك الجسم بدون تنشيف أو لفه لفوطة ناعمة قبل استخدام المرطب [لابد من استعماله على طبقة ماء]

٢- تجنب الحك بالفوطة أو استخدام (Hot air dryers)

٣- استخدام مرطب الجلد [في خلال ٣ دقايق]

باستخدام [Soakin & greasing Technique]

٤- يفضل استخدام مرطبات مثل <
- . petrolatum oint فازلين
- . fatty acids
- Linoleinic
- Prim Rose oil.
- Ceramide.

[Oint / base] فوق طبقة رقيقة من الماء

علم تعرفه: 2 Pegam- as ## ??

(2) Clothes:

- Avoid < Wool / Textile fibs
- Use < Cottons / linens (الكتان)
- Light & Not tight
- إزالة العلامات من على الملابس [اللي على الرقبة] ← irritation

→ Mild - Non alkaline cleansers

(3) Exercise: < Regular & in Warm place

(4) Enviroment: ✗ < Excess dry / " Warm

Enviroment. (Best) < Temp c 18 / Humidity 50

(5) - Avoid Contact c̄ irritants & Sensitizing Substances:-

- Antihistamines (Allergex cream)
- Neomycin
- Exacerbation of lesion on Topical Cs.

(6) Diet: →

> **Learning point** اللي تكلس xx
>
> Do not encourage cranky dieting for atopic eczema: it causes anxiety and seldom if ever does much good

(7) Health care Education: Psychological Relaxat^ & avoid Emotional stress.

Cs الأول
↓
٢ بيجامة مبلولة
↓
٢ بيجامة ناشفة فوقها

under occlusion

> **Describe the "two-pajamas treatment."**
>
> One especially effective method for applying topical anti-inflammatory medications under occlusion is known as the "two-pajamas treatment." It involves taking two pairs of cotton pajamas and soaking one pair in tepid water. At bedtime, a mild- or moderate-strength corticosteroid or calcineurin inhibitor is applied to the involved skin immediately after bathing. The wet (wrung-out) pajamas are then donned, followed by the dry pair. These are worn through the night. In the morning, this treatment can be repeated, or the patient can bathe and immediately apply emollients and clothing. This type of therapy can be modified as the "two-socks," "two-gloves," "two-caps," "two-shirts," or "two-pants" treatment as the distribution of lesions dictates.

Topical therapies

1- Cs : شروط استعمالها في حالات الـ AD :

A- Ointment :

- No ACD (less preservatives < cream)
- provide superior vapour barrier (moisurizing effect)

B. Potency :

- infants → low potent Cs (Hydrocort 1-2.5%)
- children →
  - Body → moderate potency
  - &Flexures ← Face → Elidel or weak potent Cs
  - thick LSC & lichenif →
    - daily → mild Cs
    - Weekly → superpotent.
- Monitor for growth parameters.

Weekend superpotent better > daily potent

Table 7.7 Principles of treatment with topical corticosteroids.

- Use the weakest steroid that controls the eczema effectively
- Review their use regularly; check for local and systemic side-effects
- In primary care, avoid using potent and very potent steroids for children with atopic eczema
- Be wary of repeat prescriptions

لحد ٣ أسابيع بعدها لازم اسحب تدريجيا

C use 2 Pajamas Technique :

شفوي

2 Antibiotics : may be added to Cs in cases of Acute Flare ѡ act as Antistaph & steriod sparing

oral Cephalexin

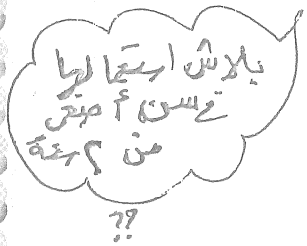
intranasal Mupiracin ont 1-3 ms

Antibs نعطي if.

- overt clinical inf.
- during Exacerbat.^

Atopic lesions Highly colonized by staph → علشان كده بيعطوا بعض Fucicort (كريمات)

③ Topical Calcineurin Inhibitors (TCI):
(Tacrolimus & Pimecrolimus)

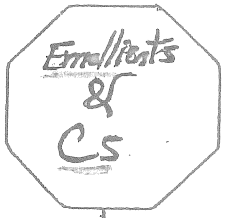


- used if there is :-
  1. Cs Resistance
  2. C.I of Cs → Face & groin to avoid atrophy
     → large surface area to avoid systemic Abs
     → Cs sensitivity
  3. Cs S.E

S.E: irritation So use Cs first then Elidel

④ Emollients :
- Petrolatum = فازلين
- 10% Urea (كريمات)
- Crude Coal Tar in 1-5% White Petrolatum.

⑤ Others :
- Classical Antiseptics
- 10% disodium Cromoglycate
- 10-30% Anhydrous Caffeine.
- Calamine lotion.



↓

Corner Stone
of
AD !!!

Table 7.8 Winning ways with emollients.

- Make sure they are applied when the skin is moist
- Prescribe plenty (at least 500 g/week for the whole skin of an adult and 250 g/week for the whole skin of a child) and ensure they are used at least 3–4 times a day
- For maximal effect, combine the use of creams, ointments, bath oils and emollient soap substitutes

Systemic therapy

الإقلاع المرجح أفضل من الفعالية → **1- Anti Histamines :-**

- Non sedating → (No) effect
- Sedating → effective by its sedating effect but (not) by its Antihistaminic effect xxx (لأنه بيساعد على النوم)

X - Canbe Combined ē:

- Ketotifen → when suspected air borne Allergy
- di-Na Cromoglycate → when suspected food Allergy.

2- PDE$_4$ inhibitors :

- X - papaverine
- X - Caffeine
- X - theophylline

→ Non specific PDE → Not effective ?? (ملوش لازمة XX)

↓

Recently

- Imidazolidinone XX
- Ro 20-1724
- CP 80 633

كريزا بورل
Crisaborole (Eucrisa)® ← Topical (FDA 2016) — Recent — المهم

Apremilast® (otezla) ← Systemic (Not yet FDA) → PS

3- Others :

- IVIG
- MTX
- Azathioprine.

Phototherapy

NB-UVB → Ttt of choice for Severe AD.

Other Ttts

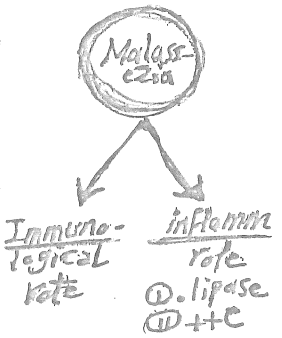
← Hypnotic, Herbal, Thy[illegible]

Endogenous Ecz.

Seborrhoeic dermatitis

def. Type of Ecz. (or papulosq. dis.) that affect the Seborrhoeic sites

. Et& pathogenesis: unknown but ± d.t. (hypersensitivity to Malassez



1-Seborrheic dermatitis is associated with normal levels of *Malassezia* but an abnormal immune response. *Malassezia* organisms are probably not the cause but are a cofactor linked to depressed Helper T cells, phytohemagglutinin and concanavalin stimulation, and antibody titers . The contribution of *Malassezia* species to seborrheic dermatitis may come from its lipase activity—releasing inflammatory free fatty acids—and from its ability to activate the alternative complement pathway. → Ros toxic effect

2- Persons prone to this dermatitis also may have a skin-barrier dysfunction (*Invest Dermatol.* Feb 2008)

↑ TG , ch
↓ Squalen , FFA

. Epidemiology : . Age : . Any but Commonest is at puberty (& peak at 40 Ys) & Neonates.

. Sex & race : (No) predilect⁻ (but males slightly > ♀).

. Clp : → . Erythematous skin & overlying greasy scales that affect Seborrheic sites (Hairy areas) as:

- Scalp
- Face → Butterfly, Nasolabial, Retroauricular, Eyelid & Lash, Eyebrow & Glabella, Ext ear, Beard & Moustach
- Upper trunk
- Flexures
- Umbilicus

"Blephanitis"

(Superficial form SD).

. clinical varieties →

→ SD may be aggravated by:

- Trauma
- Stress
- Fatigue
- Seasonal Changes & Humidity
- Drugs – Clorpromazine
  – Buspiron
  – Psoralens
- griseofulvin
- Phenothiazines (3P)

. sunlight → (1st ↑↑ then ↓ Improved)
. ↓ general Health
- HIV
- Parkinsonism
- CHF

may → Erythrodermic SD

Table 17.3 Clinical patterns of seborrhoeic dermatitis.

Infantile (Chapter 14)
Scalp (cradle cap) — cradle مهد
Trunk (including flexures and napkin area)
Leiner's disease
Non-familial → generalized SD — leinerts dsc
Familial C5 dysfunction →
Adult
Scalp
Dandruff (non inflamm.)
Inflammatory—may extend onto non-hairy areas (e.g. postauricular) (Corona Seborrheica)
Face (may include blepharitis and conjunctivitis) — Sebops → "for overlap cases"
Trunk
3P — Petaloid
Pityriasiform → psoriasiform.
2F — Flexural
Eczematous plaques
Follicular (DD - Darier)
Generalized (may be erythroderma)

Immune ↓ disorder

(d₂) effect of androgen from mother

- Seb dermatitis skin rash
- diarrhea
- FTT
- ↓ C5

fatal

. Some NBs:

(1) petaloid form (petal like): ورقة نركبة

SLE شبه

- affect presternal & interscapular areas نصف الصدر
- start as: Erythematous follicular, scaly, greasy Papules → Coalescence → figured circinate Eruptions.

(2) pityriasiform ch. By:

more → Generalized, Extensive, Slow resol.

- Generalized & more extensive > pit. Rosea.
- in particular affect Neck upto Hair lines
- resolve spont but slower pit. Rosea.

(3) Dandruff (pit. Capitis or sicca): & pit oleosa

قشرة صغير erythema

- Non inflammatory SD (1st degree of S.D)
- chr. cases of scalp dandruff & SD ± → Hair loss.

4. Severe SD may be complicated by Eczema Elsewhere:
   - Pompholyx
   - Discoid Ecz.

5. paranasal Erythema ± Flushing in Women ± D → SD or Rosacea.

* Histopathology → Non specific used to Exclude other Conditions as PS
  - Hyperkeratosis
  - Acanthosis
  - Spongiosis
  - Accentuated Rete
  - ± Neut. infilt.

Ttt of SD

(A) تعليمات المريض:

1. Ttt is for control Not cure.
   عشان كده لازم تواظب على العلاج (ممكن سنين)

2. ممنوع استعمال:
   - Alcohol based solutions xx
   - Hair tonics. xx

3. غسيل الشعر بالماء والصابون باستمرار.
   مفيد ليه
   - ✓ remove lipid
   - ✓ substrates of Malassezia.

(B) Topical Ttt:

1. Topical antifungals → as in TVC ✓ < shampoo < Zn pyrithione, Tar, Selenium sulphide; creams ↓ Ketoconazole cr

2. Topical Cs:
   شرطين لاستعمالها
   - ضعيف ← Weak (Hydrocortisone 0.5%)
   - ولمدة قليلة ← short course
     ↓
     To avoid:
     - recurrence
     - dependance & rebound.

3. other Topicals:
   - Calcineurine inhibitors
   - Imidazole 1% gel (2008) ←
   - Benzyl peroxide 5%
   - Propylene glycole.

   severe → 5% Salysilic acid cr

(C) Systemic therapy: = Syst Antifungal

1. Resistant cases →
   - Ketoconazole (1X1X14) اسبوعين
   - Itraconazole (1X1X21) ٣ اسابيع
   - ~~Lamisil~~ (غير فعال ...)

2. Generalized severe: →
   - Prednisolone 30 mg/d = syst Cs
   - Retinoids

(D) Phototherapy: UVB for resistant cases.

Stasis Dermatitis (Gravitational = Varicose ECZ.)

Def.: Stasis dermatitis is a common inflammatory skin disease that occurs on the lower extremities in patients with chronic venous insufficiency with venous hypertension.

Pathophysiology: الخطوات التالية (قراءة سريعة)

↓↓ valve competency (Disturbed function of the 1-way valvular system in the deep venous plexus of the legs).

d.t ① Aging.
② DVT
③ Surgery (stripping or saphenous V. harvesting for Coronary by Pass).
④ Traumatic injury

↓

Chronic venous insufficiency (backflow of blood from the deep venous system to the superficial venous system)

↓

Venous hypertension

↓

Stasis dermatitis

→ there are (2) Theories Explaining the mechanism by ⓦ the Venous HTN can cause cut. inflamm. of stasis Dermatitis:

1. Stasis / Hypoxia theory: stasis of Blood inside superficial venous system → Hypoxic damage of overlying skin.

stasis of Blood (Sup Veins) → Hypoxia of skin (damage)

← Not Accepted because leg Veins in those Patients were shown to have ↑ Flow rates & = not stasis
Not hypoxic = High O2 Tension (unknown Et).
بسبب هذه النظرية حدث هذه التسمية → stasis D. فالبعض يرفضها استخدام هذا المصطلح

2. Fibrin cuff theory (Dermal Microcirculation theory):-
- Venous HTN → ↑ permeability of dermal capillaries → Fibrinogen leakage → Fibrin → Fibrin Cuff formation around dermal capillaries.

Fibrin Cuffs around Dermal Vs

Barrier

Serves as barrier to O2 diffusion to skin → Tissue Hypoxia & Cell damage.

Leukocyte ← TGF B1, ICAM1, VCAM1

Traps activated Leukocytes

TGF B1 ← (1.) Release of TGFB-1 (mediator for dermal fibrosis)

ICAM1, VCAM1 ← (2.) ↑ Expression of ICAM-1 & VCAM-1 (Potent chemoattractants to keep Leukocytes in perivascular Environment).

Cut. inflamm. & Fibrosis → proteolytic enz, Free radicles → Tissue damage

(HL) → NB, 3 factors Contribute in formation of Fibrin Cuffs:-

1- ↑ permeability of Dermal Capillaries (d.t ↑ pressure)
2- ↓ Cut fibrinolytic activity
3- Leukocyte-Mediate Cytokine products

- Dermal fibrosis is the hallmark of advanced stasis D.
- Fibrin cuffs not found in ulcers d.t causes other than Venous HTN.
- Herouy et al (2001) → suggested that metalloproteinases may be important in lesional skin remodling in persons with stasis D.

- Epidemiology:
  - Incid: 6% in pts >50 yrs & 20% in those >70 y
  - Age: usually >50 yrs
  - Sex: F ± > M (Effect of preg. on Venous system)

Clp: · the medial Ankle is most frequent & severly involved area (because it represent a water shed area with relatively poor Blood Flow compared with rest of the Leg); with advancement of the dis → encircling of ankle, Below Knee Extension (Stocking Erythroderma) & ± dorsal feet.

site

lesion
- ⊙ Reddish-Brown discolorat~ is the Earliest sign.
- ⊙ there are Severe acute inflammatory, Weeping Pathes & plaques that may be ass. with: Honey-colored Crusting (d.t bact.) or Monomorphous pustules (d.t Cut. Candidiasis).
- long standing lesions may show:
  1- Hyperpigm.
  2- lichenification ?? (Hemosiderin deposit~)
  3- Lipodermatosclerosis (Dermal fibrosis with inverted champagane bottle appearance).
  4- Pseudo Kaposi Sarcoma = Acroangiodermatitis
     - Unique feature may be seen in stasis D ch' By Violaceous Nodules & plaque on dorsal feet that may undergo painful ulcerat~ simulating "Kaposi Sarcoma"

Complications:-
1- Impetiginizat~ & 2ry bact. inf
2- Cellulitis.
3- Id React~
4. lichenificat~.
5- Hyperpigm.
6- lipodermatosclerosis.
7. Atrophie blanche
8. ulcerat~ (stasis ulcer)
9- CD (sp. from HH)

× ⊙ Skin may show other venous insufficiency changes:-
- Edema
- Varicosities
- Atrophie blanche
- Hyperpigm. (d.t Hemosiderosis)

- investigations:
  1- Doppler studies → diagnosis of DVT & Valve incompetence
  2- Histopath.: [A] Acute & subacute lesions → spongiosis with superficial perivasc. infiltr.
  [B]- Chronic:
  - Acanthosis
  - Hyperkeratosis
  - deep dermal aggregates of Siderophages
  - dermal Fibrosis
  - intimal thickening of < arterioles. venules.

si Eczema + Hemosiderosis

Q NB [C] diff. bet Pseudokaposi & kaposi :

- pseudoKaposi : Typical changes of stasis D. + capillary & fibroblast prolif. → preexisting
- Kaposi : Vascular slits + Atypical endothelial cells + prolif. of vs independant of the preexisting one

(افتكر تحليل ريكوست جرعة أوعية) Treatment (2007)

Compression therapy

1. by special stockings (جراب الدوالي) that deliver controlled gradient of pressure
2. always Leg elevations

دايما بيبقى رافع رجله أثناء الجلوس أو لما ينام وحيا أو يلبس الشراب (↓ lowest leg Edema)

قبل استعمال الشراب لازم :

Assesing pt. arterial circulat° as if there is impaired circulat° → ↑ claudicat° & ± ischemic damage

Medical ttt

1. Acute Weeping ECZ → Burrow's (K permanganate 1/8000) Sol. + Mod. (ليه؟) Potent Cs (oint)
2. Chr. stasis D → calcineurin inhibitors. Why (No Atrophy & ↓ SE of Cs, No Tachyphylaxis)
3. ttt of inf : Topical & systemic Antibiotics

(CD) تعملوا Neomycin, Bacitracin ← لا يلا ش XX

4. systemic Cs : if there is systemic autoEczematizat°
5. long Term : Emollients ✓ under occlusions. الأهم

لاحظ : شروط استعمال الـ Cs هنا

- ① oint
- ② Mod. potency (super potent → systemic absorption, Atrophy → ulcer)
- ③ Not for long durat°

Surgical ttt

1. incompetent perforators → ligat°
2. Hemosiderosis → IPL (2008)

Recent ttt

- use of drugs that ↓ Neutrophil Mediated cytokines Release as:-
  1. pentoxifylline
  2. PGE1.

Dyshidrotic Eczema
(Pompholyx, Vesiculobullous hand eczema)

Def: Dyshidrotic eczema is a recurrent or chronic relapsing form of vesicular palmoplantar dermatitis of unknown etiology. Dyshidrotic eczema also is termed pompholyx, which derives from *cheiropompholyx*, which means "hand and bubble" in Greek.

Etiology and pathophysiology: The etiology of dyshidrotic eczema is unresolved and is believed to be multifactorial. Dyshidrotic eczema is considered a reaction pattern caused by various endogenous conditions and exogenous factors:

* 1- Genetic: → AD familial pattern ± present
  → Pompholyx gene in chromosome 18q22.1-3
2- Atopy (50% of cases).
3- dyshidrosis: . Not a cause but associate it in (40%) of cases & its ttt improves pompholyx. (aggravating factor)
4- Emotional stress.
5- CD (Nickel or Cobalt in diet).
6- septic focus e.g strept or dermatophytide.
* 7- Drugs: - Aspirin
  - OCPs
  - IVIG
* 8- UVA phototherapy (2004)

may be reaction to fungus infection of feet = Trichophytid

NB: DD ① PP pustular Ps. → the (1ry) lesion is pustules (not clear vesicles)

* ② chr. Vesiculobullous Hand Ecz: usually (no) remission & the vesicles always present.

. Epidemiology :-

- 3rd most common cause of Hand ECZ.
- usually affect middle Aged (≃38 Ys)
- (M = F.)

. C/P : Acute onset of Eruption of Macroscopic (bilat غالبا) Tapioca like deep seated vesicles & bullae on NL background on Both Palms & soles & sides of Fingers. (مكان)

(Nail fold ± affected → Nail dystrophy.)

- [Commonly there are ass :-]
  ("شديد") → [pruritus : may precede the Eruptn.
  [Hyperhidrosis
- [Site] : [palms only → 80%. (Called cheiropompholyx).
  [Sole " → 10%. (" Pedo ").
  [Palm & Sole → 10%.

- [Fate] : outbreaks Resolve spontaneous over several ws (≥ 2-3ws) & Recurrance is common (U.) at intervals of ≃3-4 ws for ms - Ys.

DD = (NB) - (uncommon unilat. cases) → (suspect CD.)
(→ CD → unilat; ch vesiculobullous dermatitis على طوله موجودة)
- (Pompholyx) should be reserved for Typical cases in ̄w attacks resolve & recure while (chr.) recurrent vesiculatn without remission called chr. vesicular dermatitis.

. Histopathology : - Spongiosis ̄c epid. Lymphocytic infilt.
- intraepidermal vesiculatn

. ttt : [1] remove possible causes:
[• stress
[• Hyperhidrosis
[• CD
[• Fungal inf

hyperhydrosis ttt
اللونسوم أربعتاني مرة
فيما ستريك لمدة 10 دقيقة
Al. acetate

[2] acute cases → Drying antiseptic lotn ̄c draining of large bullae
[3] subacute & chr. cases → Cs oint → Emolients & Keratolytics
[4] Resistant cases → MTX & radiatn ttt → photochemo therapy.

12

= Fissuring

Ref. All Sources

Asteatotic Eczema
Eczema craquelé, Xerotic eczema (Chapping)

Def. Eczema Characterized by pruritic, dry, cracked, and polygonally fissured skin with irregular scaling . It most commonly occurs on the shins of elderly patients, but it may occur on the hands and the trunk.

Etiology and pathophysiology:

Causes

↓lipids & water of skin

↓ NMF Natural Moisturing Factor

***Multiple etiologic factors may coexist to cause asteatotic dermatitis, including the following:** *(All are associated with ↓↓lipid content of skin):*

- **Aging:** due to ↓↓sebaceous and sweat glands activity and ↓↓ Keratin synthesis.
- **↓humidity and cold:**→ increase the loss of water by convection.
- **Wrong Behaviour** :-Frequent or prolonged bathing in hot water and use of soaps, infrequent use of emollients and use of Degreasing agents (Solvents and Cleansers)

- **Atopy**
- **Ichthyosis**
- **Radiation**
- **Drugs** - Antiandrogen therapy[5] and diuretic therapy
- **malabsorption and Nutritional deficiencies** of essential fatty acids, including linoleic acid and linolenic acid , Zinc deficiency[3]
- **Thyroid disease** - Myxedema and other thyroid diseases with diminished sweat and sebaceous gland activity[4]
- **Neurologic disorders** - Decreased sweating in denervated areas
- **Malignancies** - Malignant lymphoma,[6] gastric adenocarcinoma,[7] glucagonoma, angioimmunoblastic lymphadenopathy,[8] breast cancer, large-cell lung carcinoma, and colorectal carcinoma[9]

Epidemiology: *Age: elderly (>60 y.) *Sex: (M>F)

c/p: (Primary lesions:) Slightly scaly, inflamed, curvilinearly cracked and/or fissured skin most commonly involves the pretibial areas, but it may also occur on the thighs, on the hands, and on the trunk *(Fitzpatrick likened asteatotic eczema to a dried-up riverbed).*

(Secondary lesions:) Excoriated, erythematous, edematous patches may result from rubbing or scratching.

Clinical types: 1-localized: usually (pretibial)

2- Generalized (Ichthyosis): ?? Mg

III

Soaking and Greasing :-
(Soak & Smear) Technique

Soaking : of affected part in Tepid Water for 1/4 min

Greasing (Smear): Immediate use of mild Cs oint

1 تعليمات للمريض : الحمام بتاعك لازم :

١- لفترة قصيرة (=١٠ دقايق)
٢- بماية فاترة
٣- بلاش صابون
٤- بلاش صوف
٥- استخدم Humidifier

2 Emoliants:
- استعماله بعد الحمام مباشرة (See AD)
- استعماله يوميا : ٢-٤ مرات على الأقل

3 Topical Cs :
Mid Topical Cs (III-VI)
يمكننا استعمالها لطريقة
مرة اثنين

A Soaking & Greasing B Occlusion For 1-2 ds

60

Nummular Eczema
(Discoid Ecz.) (14)

Def. Type of Endogenous Ecz. ch BY: (!!!)
well defined, Coin-shapped, scaly, plaques usually on arms & legs. w̄ are → very itchy & → Very persistent

- AET & pathophysiology :-

Atopy (1-) AD (recently considered as Adult onset AD)

(2.) Infect= (staph usually colonize or infect it).

(3-) Emotional stress.

(4-) local & physical Trauma: عملت تعبير

- Insect bite
- Abrasions
- CD

dry (5.) Xerosis → disturbed barrier → ↑ Penetrat= of Allergens → CD.

(6-) Alcohol Abuse.

Drugs = (7) Generalized form Recently ass ē:

- IFN ℞ for hepatitis C
- Ribavirin.
- Anti TNFα.

(8-) Autoeczemati sat=n (Discoid Ecz ملتها بها)

C/P: (1) Age: 2 peaks

- (60-70y) (++)
- (20-30ys) (+)

(5)

- C/P :- Well demarcated Coin shaped plaques of Closely set, thin Walled Vesicles on an Erythematous base ⓦ are dull red, oozy → this may be followed by Central Clearing & Peripheral Extension → ring shaped or annular lesions may Fade → dry scaly Patches.

pid

Clinically: Very itchy & Very persistent.

- after any period bet lads - ms, (2ry) lesions occur often in a mirror image Configuration on opposite side of Body.
- a very chic feature of Ecz. that the patches ⓦ have apparently become dormant may become active again specially if ttt is discontinued prematurely.

- X. 3 patterns are recognized :-
  1. Discoid Ecz. of hands & forearm
  2. " " " Limbs & Trunk
  3. Dry Discoid Ecz.

- Type 1 → dorsal Hands & Finger sides
- Type 2 → seen in elderly & pts. c Xerosis.
- Type 3 → Dry discoid plaques on legs & arms, mild itchy (Dry Skin) & resistant to ttt

- Course Waxes & Wanes for Years with recurrence in cold or dry climates

---

Is there a cure for nummular dermatitis?
No. However, the disease can be controlled. Many of the same principles apply here that apply to the treatment of atopic dermatitis. Limiting baths and soap exposure, avoiding irritants, frequent use of emollients, topical corticosteroids, avoiding dry environments, and antihistamines all have a role in treatment. Topical corticosteroids are the mainstay of therapy. With the high rate of staphylococcal colonization, many dermatologists routinely prescribe a 2-week course of oral antibiotics, such as dicloxacillin or cephalexin. Systemic steroids should be used only for severe cases and limited to a tapered course over 2–3 weeks. Severe chronic cases may also benefit from PUVA.

(ttt)

Staph Carrier also

Waxes & wanes

7. Does nummular dermatitis resolve spontaneously?
Yes, but not often. In a prospective study of patients followed for 2 years, 22% were disease-free. Another 25% were free of lesions for weeks to months, but 53% were free of lesions only with continued local therapy. If there is no clearing within 1 year, the disease tends to persist for many years.

Juvenile planter Dermatosis

(. DNNZ)
. Andrew
. Rook

(18)

def Eczema affecting the feet of children.

AEt: . Repeated friction & Macerat^n of feet by: "الكوتشي"

[1]. occlusive shoes specially Asthletic Shoes

[2]. Abrasive effect of pool surfaces or diving board (ترنب)

[3]. (Thin) Non-absorbant synthetic stocks.

[4] Some consider it manifs of AD.

. تعتقد ان سببها هو التغير اللى حصل فى آخر ٣٠ سنة فى صناعة الأحذية وشرابات الأطفال
حيث تم استبدال المواد الطبيعية (الصوف، القطن، الجلد) بمواد صناعية (نايلون وبلاستيك)
والتى تعتبر أقل مسامية من المواد الطبيعية بالإضافة الى تغطية الأحذية بمواد repellent
علشان تحسن الـ durability بتاعة سطح الحذاء.

[all these]: ↑ Humidity → Feet macerat^n → sweat retent^n → aggravate the condition

ليست دة هى كل القصة لانه لوحظ فى الأطفال اللى بيلبس صندل جلد بيحصل برضه
(So role of friction considered) ✓

CIP [1] Age: (3 Ys) – (puberty) (14).

[2] start as bilat, symmetrical red glazed smooth patches at medial or dorsal Aspect of (Big Toe) → then spread to involve weight bearing areas of feet as forefoot. (Heel less affected).

[Note]: Webs & arches are spared.

DD: [AD. / CD] [psoriasis / Fungal inf.]

Treatment: (1) Reduce friction: جزمة جلد – شفابة للقدم – شرابات قطن (porous foot wear).
(2) Emollient.
(3) Have rest day: schedule quite time ē Little or No walking to allow for Healing of fissure
(4) Topical Cs

↓ ممكن تختفى (لوحدها عند البلوغ)

Q

Hand eczema

تحريري

Hand eczema is such a common and distressing condition, and poses such difficult problems for the dermatologist, that it deserves separate consideration . Up to 30% of occupational medical practice relates to hand eczema, with important issues regarding medical litigation, worker's compensation and disability. One-quarter of the patients referred to a specialized contact dermatitis clinic suffered from hand dermatitis.

Classification: 1-Etiologic classification (Rook) 2-Morphologic classification

3- Classification Acc. to the Age.

1-Etiologic classification

Exogenous	*Endogenous*
1-ACD: •Delayed hypersensitivity (type IV) (e.g. chromium, rubber) •Immediate hypersensitivity (type I) (e.g. seafood) 2- ICD: •Chemical *(e.g. soap, detergents, solvents)* •Physical *(e.g. friction, minor trauma, cold dry air)* 3-Ingested allergens *(e.g. drugs, possibly nickel, chromium)* 4-Infection *(e.g. following bacterial infection of hand wounds)* 5-Secondary dissemination *(e.g. dermatophytide reaction to tinea pedis)*	1- Atopic 2- Dyshidrotic (Pompholyx) 3. Psychosomatic (↑↑ Eczema > Initiation) → aggrevate ecz 4- Idiopathic (Discoid & Hyperkeratotic Palmar Ecz).

Most common exogenous Cause →

2-Morphologic classification

1-vesiculobullous hand eczema(Pompholyx) / & Patchy vesiculosquamous
2-hyperkeratotic hand eczema / 7
3-dry palmer eczema = House wives Ecz = Wear & tear dermatitis
4-finger tip eczema
5-ring eczema ⑥ Localized thumb Ecz
7-discoid eczema
8-chronic acral dermatitis
9-apron eczema
10-gut eczema
11-other patterns (eg.patchy vesiculosquamous)

→ Recurrent Not itchy | No vesicles | Local Palmar Peeling.

1- Chr. Vesiculobullous Hand Ecz: See Pompholyx.

(2) Hyperkeratotic Hand Ecz. (Tylotic Ecz)

- Unknown Etiology
- (M) > F usually middle aged & over
- Clp → highly irritable Scaly Fissured, Hyperkeratotic Patches on palms & palmar aspects of fingers.
- DD: Palmar Ps → "ch" By:
  - silvery scales
  - Sharply demarcated Scalloped edges to the Erythema along borders of < Hands & Fingers.
  - xx Absence of pruritus xx
  - N ↙ involvement of Knuckles.
  - N ↙ Nail pitting (±out Nail fold affects)
  - other psoriatic sites.
- ttt: (Extremely refractory to ttt):
  - Cs + Keratolytics
  - PUVA
  - Etretinate.
  - Grenz Zone ttt

(3) Dry palmar Ecz = House wives Ecz = Wear & tear dermatitis:

→ usually affect House wives & cleaners d.t contact with water, detergents, soaps, solvents & even Food Cuttings.

- Scaly
- Dry
- Criss Cross
- cracks
- lack of pliability
- ± chapping

- The skin looks Dry, criss crossed ē superficial Cracks ass. with damaged Horny layer & unable to respond ē its Normal pliability to hand & finger movement.
- there may be ass. with dorsal Hand chapping

(4) Finger-Tip Ecz:

- affect palmar aspect of finger tips usually localized but may extend to the palms.

clp - Finger tips are → dry, cracked, painful & Fissured.

+ There are (2 Types) Finger Tip ECZ

Type I (Common)
affect most of all fingers of dominant hand specially Thumb & forefingers
↓ its d.t
(ICD) (Cumulative)

Type II (less common)
affects thumb, forefinger & ring finger of dominant hand → usually occupational
↓ ± d.t
ICD
ACD

(5) Ring ECZ

→ accomulation of solvent's & soaps under Rings

- Cause: يتجمل تحت الدبلة أو الخاتم
نتيجة تراكم الصابون والمواد المنظفة وليست
حساسية للمادة الدبلة أو الخاتم
- site: Wedding ring finger & may extend to middle finger

(6) Localized Thumb ECZ

- child who put fingers in mouth.

في الأطفال الصغار الذين يضعون الأصابع
في الفم باستمرار

- may be (asse) → Nail dystrophy

(7) Discoid ECZ → see before

(8) Chr. Acral dermatitis:-

ECZ + ↑IgE → ↓↓ Cs systemic

- distinctive synd. ch. By chr. hyperkeratotic papulo-Vesicular ECZ of Hands & feet intensly pruritic & is ass. with marked ↑ IgE in middle Aged (without) personal or FH of Atopy
- (↓↓) → Cs (systemic); Topical (not) effective

XX

(9) Apron Ecz : Ecz caused by ICD affects palmar aspects of adjacent fingers & forming in a circular pattern over MCP in a pattern resembling Apron.

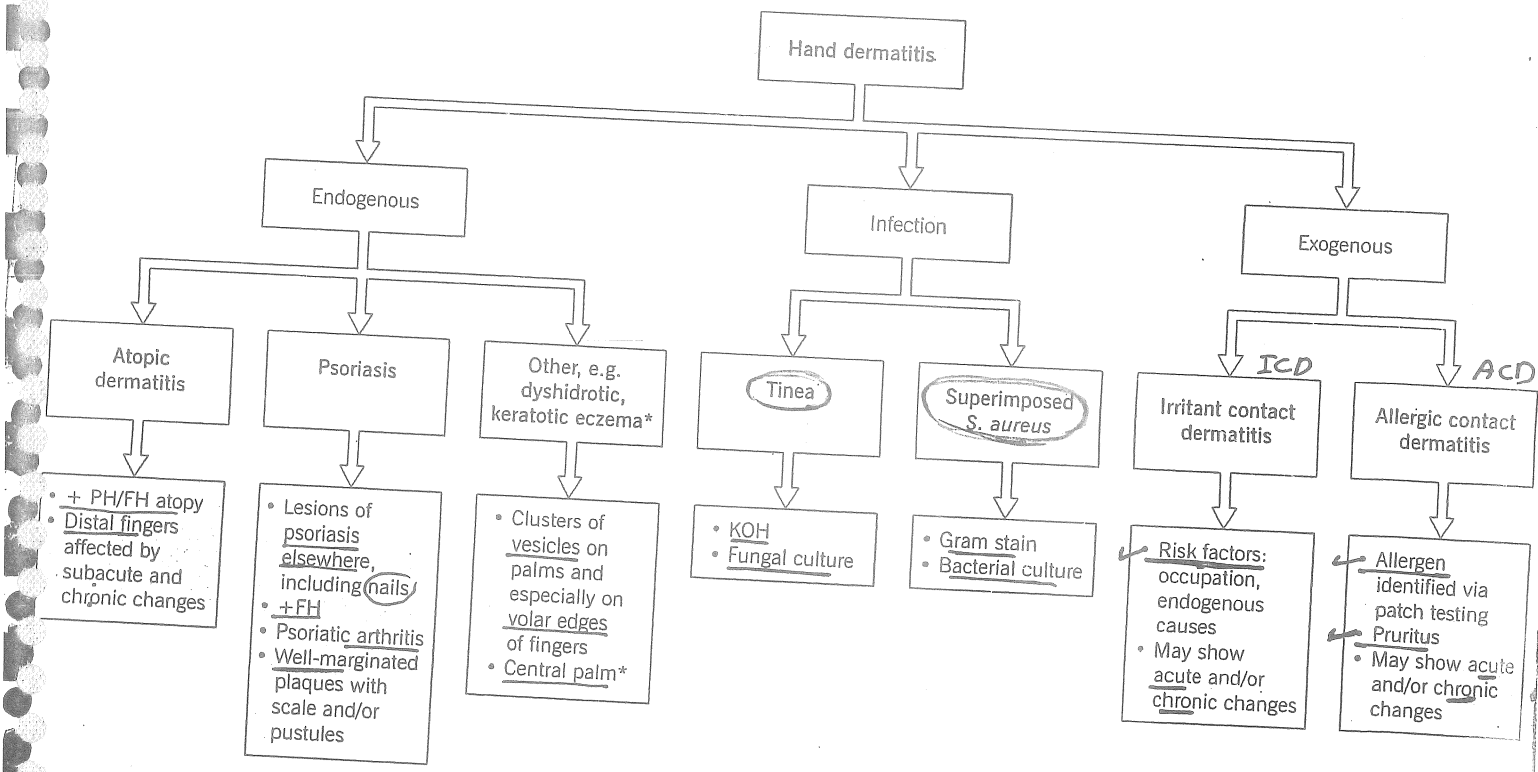
(10) Gut Ecz (Slaughter house Ecz):

- This type of Ecz affect workers who eviscerate & clean pig carcasses
- Clp: Vesicular Ecz starts at fingerwebs & spreads to sides of fingers.

→ usually self limiting & clears in ≃ 1-2 ws but may recur

- Et: unknown but ± d.t fat or blood ICD

(11) Patchy Vesiculosquamous Ecz : Bilat, Asymm. mixture of irregular, patchy, vesiculosquamous lesion



* some clinicians view keratotic eczema as a form of psoriasis

Classification of hand dermatitis. More than one etiology may be present, e.g. atopic dermatitis plus irritant contact dermatitis. FH, family history; history. *Courtesy, David E. Cohen, MD.*

Lichenification

Introduction

Lichenification is a pattern of skin response to repeated scratching or rubbing, characterized *histologically* by acanthosis, hyperkeratosis, and elongated rete ridges, and *clinically* by a thickened skin, with accentuation of the surface makings so that the affected skin surface resembles *tree bark*. It may be primary (lichen simplex), without an itchy skin disease and caused by emotional tension, or secondary to an itchy skin disease as venous eczema, atopic dermatitis, chronic contact dermatitis, or chronic infection with T. rubrum of thighs or feet.

& hyperpigm

Lichen simplex chronicus (LSC)
(localized Neurodermatitis)

Def. React^ pattern of skin that arises (2ry) to repeated scratching [or] rubbing [so this (not) a 1ry process] & ch^ by cut. lichenificat^ Δ of:

Δ
- Thickening
- Hyperpigm.
- Accentuated skin Mark ings.

· Etiopathogenesis (unknown but ±:)

↓

• Emotional stress → Sensat^ of (Burning or Pruritus) → rubbing → lichenificat^ → More rubbing → More lichenific.
(Viscious Circle of itch/scratch Cycle)

Epidemiology: [Age]: any but usually (30-50ys)
[Sex]: (♀) > ♂

· C/P (1.) itching: · Severe & occurs in paroxysms of great intensity → full scoring.
· there is (refractory period) of some hours before until itching recur.

(2.) Skin lesion: - [at first]: Erythematous, Edematous Excoriated plagues → classical Picture of lichenificat^
- Lichenified papules ± Pecn.

• Site: Most Common sites are:

Nape Neck → [occiput
Nuchal area (Lichen Nuchae)] ♀
– Perineum & Scrotum ♂
[Wrist & ankle
Extensor forarms

NB: (i) Giant lichenification of Pautrier
LSC in areas of loose S.C.T as "Genito-Crural area → Solid Tm like plaque ē Warty Cribriform surface.

(ii) Notalgia Paresthetica LSC at inf. tip of Scapula.

• Pathology → see lichenification

• DD (1) lichen amyloidosis (2) L-p (3) Ps.

• treatment:-

1- Stop itching (Break the itch/scratch cycle):-
[Anxiolytic
مهدئات

2- Cs: Topical & ILs

3- Emollients

4- Antihistamines, Botox (2009), Topical Aspirin/dichloromethane (JAAD 2001)

5- TENS (Transcut Electric N. Stim).

Other Types of Eczema

- Frictional lichenoid Dermatitis
- Eye-lid Ecz.
- Breast Ecz.

• Sandbox or Frictional lichenoid Dermatitis.

- Pin-head sized, White papules at friction sites
- Etiopath. ?? friction or Sun.
- DD: lichen-nitidus.

(NB) In Adults: Dermatitis papu... Juvenilis

2- Eye-lid Ecz. →

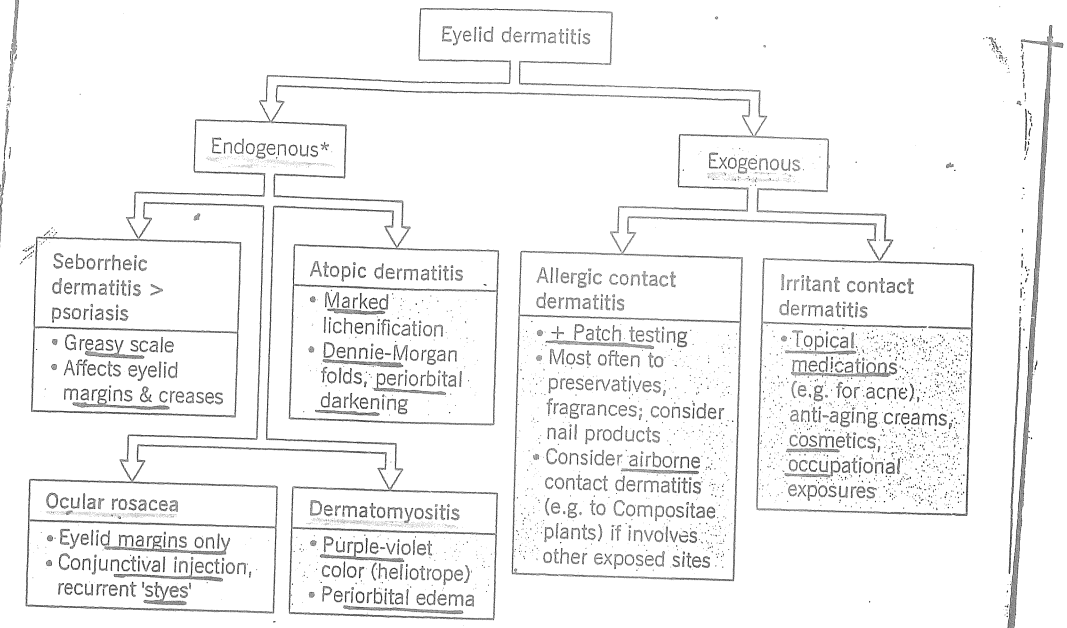


Fig. 13.6 Classification of eyelid dermatitis. More than one etiology may be present, e.g. atopic dermatitis plus irritant contact dermatitis.

3- Breast Eczema (Nipple Ecz.)

- ± Affect Nipple, Areola & surrounding skin. (Specially of Nursing Mother).
- Etiology: CD, ND, AD, SD
- ttt: As Ecz. → if (No) response → (Biopsy) to Exclude "Mammary Pagets" ??
  - Unilat.
  - Not responding to (CS)

Acroangiodermatitis

(Synonyms: pseudo-Kaposi's sarcoma, acroangiodermatitis of Mali-Kuiper, gravitational purpura, stasis purpura)

- Definition: was first coined by Mali in 1965. [1] It is a proliferation of *pre-existing vasculature* seen in venous hypertension, arteriovenous malformation, or acquired iatrogenic arteriovenous (AV) fistula.
- Ethopath. : chr. Venous insuff → Venous HTN → Tissue Hypoxia ← Neovascularization + fibroblast prolif
- C/P: Confluent, violaceous or brown-black papules cover large areas of the distal parts of the legs. Ulceration and bleeding are sometimes noted. Bilateral lesions are usually associated with chronic venous insufficiency, whereas unilateral lesions suggest an underlying vascular malformation.

- Types:

1. Mali Type → ass. with Stasis Dermatitis
2. Stewart-Bluefarb Type → ass. with Cong AV malformation
   e.g. Klippel Trenaulay synd
3. Dermite ocre of Favre: ass. with pregnancy.
4. other Types:
   - ① AV Fistula of ← shunt in CRF / stump Dermatosis in Amputees
   - ② HCV ass. (EMed. 2010).

	Acroangiodermatitis of Mali	Kaposi's sarcoma
Hp	Small dilated vessels lined by plump endothelial cells with hyperplasia of pre-existing vasculature	Slit-like spaces and spindle cell proliferation. Vascular proliferation is independent of the existing vessels
PAS	+ve	-ve
Factor VIII (B) Ag in Endoth	+ve	-ve
CD34	Positivity seen on endothelial cells of hyperplastic vessels	Positivity seen on both endothelial cells and the characteristic spindle-shaped, perivascular cells
RBCs extrav Hemosiderin & Fibs	Present	Present

Treatment

1- Correction of the underlying chronic venous insufficiency and vascular malformations

2- *Systemic therapy:* Various medical modalities of therapy have been tried with favorable results but options are limited. Oral erythromycin 500 mg four times a day (or) Dapsone

3- Topical Cs

4- Vascular lasers.

Dyshidrotic Eczema
(Pompholyx, Vesiculobullous hand eczema)

Def: Dyshidrotic eczema is a recurrent or chronic relapsing form of vesicular palmoplantar dermatitis of unknown etiology. Dyshidrotic eczema also is termed pompholyx, which derives from *cheiropompholyx*, which means "hand and bubble" in Greek.

Etiology and pathophysiology: The etiology of dyshidrotic eczema is unresolved and is believed to be multifactorial. Dyshidrotic eczema is considered a reaction pattern caused by various endogenous conditions and exogenous factors:

* 1- Genetic: → (AD) familial pattern ± present
  → (Pompholyx gene) in Chromosome 18q12.1-3
2. Atopy (60% of cases).
3. dyshidrosis: Not a cause but associate it in 40% of cases & its ## improves pomphlyx.
4. Emotional Stress.
5. CD (Nickel or Cobalt in diet)
6. Septic focus e.g Strept or dermatophytide.
* 7- Drugs: Aspirin, IVIG & PUVA.

- Clp: Acute onset of eruptn of Bilat & symmetrical Deep seated Vesicles & Bullae at palms (Cheiropompholyx) Soles (Pedopompholyx) or Both. Nail fold may be affected → Nail dystrophy. ass. ē severe itching & Burning.
- Exacerbatn & Remission is Common ((Chronic Vesiculo-Bullous Eczema)) موجود في طول
- Unilat. cases may be d.t (CD)
- 3rd Most Common Type of Hand Ecz., usally affect middle Aged.

1- treat the Underlying cause.
2- Vesiculo- lesion → drying Antiseptic lotn
3- chronic " → Cs, Emollient & Keratolytic
4- Resistant Cases: - systemic Cs
- MTX
- Radiatn

My lesion is pustules ↑

DD: pustular psoria
لازم تفرق بين

Nummular Eczema
(Discoid Ecz.)

(1-A)

Def: Type of Endogenous Ecz ch BY:
well defined, Coin-shapped, scaly, plaques
usually on arms & legs w are very itchy &
[Very persistent

- AET & pathophysiology :-

"Atopy" (1-) AD (recently considered as Adult onset AD)
(2-) Infects (staph (usually) colonize or infect it).
(3-) Emotional stress.
(4) Xerosis

- C/p : Discoid plaques ch BY:
  - May undergo central clearing → Annuler lesion.
  - Very itchy, very chronic
  - usually at: forearms, legs, Hands, Lumb & Trunk
  - staph has a Marked Role → AB أعطيه
  - More in Elderly (60-70ys)

(##)

Is there a cure for nummular dermatitis?
No. However, the disease can be controlled. Many of the same principles apply here that apply to the treatment of atopic dermatitis (Limiting baths and soap exposure, avoiding irritants, frequent use of emollients, topical corticosteroids, avoiding dry environments, and antihistamines all have a role in treatment. Topical corticosteroids are the mainstay of therapy. With the high rate of staphylococcal colonization, many dermatologists routinely prescribe a 2-week course of oral antibiotics such as dicloxacillin or cephalexin. Systemic steroids should be used only for severe cases and limited to a tapered course over 2–3 weeks. Severe chronic cases may also benefit from PUVA.

← قواعد عامة
← staph Carrier فيه
ABs
Topical Cs
emollients
Antihistamine
Syst Cs
PUVA

(Rate)

Does nummular dermatitis resolve spontaneously?
Yes, but not often. In a prospective study of patients followed for 2 years, 22% were disease free. Another 25% were free of lesions for weeks to months, but 53% were free of lesions only with continued local therapy. If there is no clearing within 1 year, the disease tends to persist for many years.

(EMal 2009) 10

Pityriasis Alba

def: Non specific dermatitis of unknown Aetiology that causes Erythematous scaly patches → these resolve & leave areas of Hypopigm that slowly repigment to NL.

Aetiology: unknown but ± d.t:

assé
1. Atopy
2. strept. inf.
3. Sun Exposure (أهم حاجة)
4. ↓ Zinc.
5. ↓ Fe
6. Parasitic infestations.

7- Malassezia. produces a substance called pityriacitrin → sun filtrat° (prevent Natural sun Tanning).

Topical # Antifungal
كلام عام

C/P:
1. usually affects dark skin children at summer
2. has ② stages: مهم
   - Early: ill defined pink erythematous, scaly lesions →
   - Late: Hypopigmentat° (ill defined & scaly)
3. usually at sun exposed areas
4. Clinical Varities:
   - (i) classical Type: at sun exposed areas
   - (ii) Generalized " = Bilat & symm.
   - (iii) pigmenting " : Central bluish pigmentat° surrounded by ill defined slightly scaly Halo at the face

Treatment:
صعب جداً وبيرجع بسرعة

1. Treating the cause e.g sunscreen, parasites, vit. deficiency
2. - Early stage → Hydrocortisone 1%
   - Late " → Emollient
3. Some Authers: بيعطوا لوشنات اكيلازين Ezalline (Similar to Vitilligo ttt) → SE hyperpig

(19)

Hand eczema

تحريري

Hand eczema is such a common and distressing condition, and poses such difficult problems for the dermatologist, that it deserves separate consideration . Up to 30% of occupational medical practice relates to hand eczema, with important issues regarding medical litigation, worker's compensation and disability. One-quarter of the patients referred to a specialized contact dermatitis clinic suffered from hand dermatitis.

Classification: 1-Etiologic classification 2-Morphologic classification
(Book) 3- Classificat° Acc. to the Age

1-Etiologic classification

Exogenous	Endogenous
1-ACD: -Delayed hypersensitivity (type IV) (e.g. chromium, rubber) -Immediate hypersensitivity (type I) (e.g. seafood) 2- ICD: -Chemical *(e.g. soap, detergents, solvents)* -Physical *(e.g. friction, minor trauma, cold dry air)* 3-Ingested allergens *(e.g. drugs, possibly nickel, chromium)* 4-Infection *(e.g. following bacterial infection of hand wounds)* 5-Secondary dissemination *(e.g. dermatophytide reaction to tinea pedis)* (3I — Id)	1- Atopic 2- Dyshidrotic (Pompholyx) 3- Psychosomatic (↑↑ Eczema > Initiation) زودها 4- Idiopathic (Discoid & Hyperkeratotic palmar ECZ.)

2-Morphologic classification

1-vesiculobullous hand eczema(Pompholyx) / & patchy vesiculosq.
2-hyperkeratotic hand eczema (Tylotic ECZ.)
3-dry palmer eczema (House wife Ecz)
4-finger tip eczema
5-ring eczema (تراكم المنظفات تحت الدبلة او الخاتم (وليس من الخاتم نفسه))
6. localized thumb ECZ (الاطفال بيمصوا صوابعهم)
7-discoid eczema
8-chronic acral dermatitis (Hand ECZ + ↑ IgE)
9-apron eczema (زي المريلة بتاعة العمليات)
10-gut eczema (جزارين) → blood, fat (ICD)
11-other patterns (eg.patchy vesiculosquamous)

• Treatment:
1. جوانتي باستمرار مع الغسيل وتقطيع الأكل - وصابون شوربوله
2. Potent or superpotent Cs for 2-3 ws then Week-end
3. ± 5 ds / W → Weak potent Cs ~ Tacrolimus.
4. Emollients: عدة مرات يوميا باستمرار لا يتوقف